Residual Risk Assessment for the Pulp & Paper Source Category

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AERMOD	American Meteorological Society/EPA Regulatory Model
AEGL	Acute exposure guideline level
ASTDR	US Agency for Toxic Substances and Disease Registry
CalEPA	California Environmental Agency
CTE	Central Tendency Estimate
ERPG	Emergency Response Planning Guideline
HAP	Hazardous Air Pollutant
HEM	Human Exposure Model
HI	Hazard index
HQ	Hazard quotient
IRIS	Integrated Risk Information System
MACT	Maximum Achievable Control Technology
MIR	Maximum Individual Risk
MOA	Mode of action
NAC	National Advisory Committee
NAAQS	National Ambient Air Quality Standard
NATA	National Air Toxics Assessment
NEI	National Emissions Inventory
NPRM	Notice of Proposed Rulemaking
PB-HAP	Persistent and Bioaccumulative - HAP
POM	Polycyclic organic matter
REL	Reference exposure level
RfC	Reference concentration
RfD	Reference dose
RME	Reasonable Maximum Exposure
RTR	Risk and Technology
TOSHI	Target-organ-specific hazard index
URE	Unit risk estimate

1 Introduction

Section 112 of the Clean Air Act (CAA) establishes a two-stage regulatory process for addressing emissions of hazardous air pollutants (HAPs) from stationary sources. In the first stage, section 112(d) requires the Environmental Protection Agency (EPA, or the Agency) to develop technology-based standards for categories of sources (e.g., petroleum refineries, pulp and paper mills, etc.) [*1*]. Under section 112(d)(6), EPA must review each of these technology-based standards at least every eight years and revise a standard, as necessary, "taking into account developments in practices, processes and control technologies." In the second stage, EPA is required under section 112(f)(2) to assess the health and environmental risks that remain after implementation of the MACT standards. If additional risk reductions are necessary to protect public health with an ample margin of safety or to prevent an adverse environmental effect, EPA must develop standards to address these remaining risks. This second stage of the regulatory process is known as the residual risk stage. For each source category for which EPA issued MACT standards, the residual risk stage must be completed within eight years of promulgation of the initial technology-based standard.

In December of 2006 we consulted with a panel from the EPA's Science Advisory Board (SAB) on the "Risk and Technology Review (RTR) Assessment Plan" and in June of 2007, we received a letter with the results of that consultation. Subsequent to the consultation, in June of 2009 a meeting was held with an SAB panel for a formal peer review of the "Risk and Technology Review (RTR) Assessment Methodologies" [*2*]. We received the final SAB report on this review in May of 2010 [*3*]. Where appropriate, we have responded to the key messages from this review in developing our current risk assessments and we will be continuing our efforts to improve our assessments by incorporating updates based on the SAB recommendations as they are developed and become available. Our responses to the key recommendations of the SAB are outlined in a memo entitled, "EPA's Actions in Response to Key Recommendations from the SAB Review of RTR Risk Assessment Methodologies" [*4*].

This document contains the methods and the results of baseline risk assessments (i.e., after the implementation of the respective MACT standards) performed for the pulp and paper source category. The methods discussion includes descriptions of the methods used to develop refined estimates of chronic inhalation exposures and human health risks for cancer and noncancer endpoints, as well as descriptions of the methods used to screen for acute health risks, chronic non-inhalation health risks, and adverse environmental effects. Since the screening assessments indicated low potential for chronic non-inhalation health effects or environmental impacts, including effects to threatened and endangered species, no further refinement of these assessments was performed.

2 Methods

2.1 Emissions and source data

Data from a CAA section 114 information collection request (ICR) were used for this assessment. In February 2011, we issued an ICR to all U.S. pulp and paper manufacturers to gather information needed to conduct the technology review and residual risk requirements of the CAA. The ICR requested available information regarding process equipment, control devices, pulp and paper production, bleaching, inventory data for all pulp and paper point and fugitive emission, practices used to control fugitive emissions, and other aspects of facility operations, including stack parameters and locations. Next, EPA engineers who have extensive knowledge of the characteristics of this industry performed an engineering review and thorough QA/QC of the data to identify limitations and issues. Finally, EPA engineers contacted facility and industry representatives to clarify details and resolve issues with their ICR data submissions. Details on the development of the emissions and source data for this source category are discussed in a memorandum entitled, *Inputs to the Pulp and Paper Industry October 2011 Residual Risk Modeling*, available in the docket for this rule making. Section 3 below provides a summary of the emissions.

2.2 Dispersion modeling for inhalation exposure assessment

Both long- and short-term inhalation exposure concentrations and associated health risk from each facility in the source category of interest were estimated using the Human Exposure Model in combination with the American Meteorological Society/EPA Regulatory Model (AERMOD) dispersion modeling system (HEM3). The approach used in applying this modeling system is outlined below, and further details are provided in Appendix 1. The HEM3 performs three main operations: atmospheric dispersion modeling, estimation of individual human exposures and health risks, and estimation of population risks. This section focuses on the dispersion modeling component. The exposure and risk characterization components are discussed in other subsections of Sections 2 and 3.

The dispersion model in the HEM3 system, AERMOD version 11103, is a state-of-the-science Gaussian plume dispersion model that is preferred by EPA for modeling point, area, and volume sources of continuous air emissions from facility applications [*5*]. Further details on AERMOD can be found in the AERMOD Users Guide [*6*]. The model is used to develop annual average ambient concentrations through the simulation of hour-by-hour dispersion from the emission sources into the surrounding atmosphere. Hourly emission rates used for this simulation are generated by evenly dividing the total annual emission rate from the inventory into the 8,760 hours of the year.

The first step in the application of the HEM3 modeling system is to predict ambient concentrations at locations of interest. The AERMOD model options employed are summarized in Table 2.2-1 and are discussed further below.

Table 2.2-1 AERMOD version 09292 model options for RTR modeling

Modeling Option	*Selected Parameter for chronic exposure*
Type of calculations	Hourly Ambient Concentration
Source type	Point, area represented as pseudo point source
Receptor orientation	Polar (13 rings and 16 radials) Discrete (census block centroids) and user-supplied receptors
Terrain characterization	Actual from USGS 1-degree DEM data
Building downwash	Not Included
Plume deposition/depletion	Not Included
Urban source option	No
Meteorology	1 year representative NWS from nearest site (over 200 stations)

In HEM3, meteorological data are ordinarily selected from a list of over 200 National Weather Service (NWS) surface observation stations across the continental United States, Alaska, Hawaii, and Puerto Rico. In most cases the nearest station is selected as representative of the conditions at the subject facility. Ideally, when considering off-site meteorological data most site-specific dispersion modeling efforts will employ up to five years of data to capture variability in weather patterns from year to year. However, because we had an insufficient number of appropriately formatted model input files derived from available meteorological data, we modeled only a single year, typically 1991. While the selection of a single year may result in under-prediction of long-term ambient levels at some locations, likewise it may result in over-prediction at others. For each facility identified by its characteristic latitude and longitude coordinates, the closest meteorological station was used in the dispersion modeling. The average distance between a modeled facility and the applicable meteorological station was 40 miles (72 km). Appendix 2 (*Meteorological Data Processing Using AERMET for HEM3*) provides a complete listing of stations and assumptions along with further details used in processing the data through AERMET. The sensitivity of model results to the selection of the nearest weather station and the use of one year of meteorological data is discussed in "Risk and Technology Review (RTR) Risk Assessment Methodologies" [*2*].

The HEM3 system estimates ambient concentrations at the geographic centroids of census blocks (using the 2000 Census), and at other receptor locations that can be specified by the user. The model accounts for the effects of multiple facilities when estimating concentration impacts at each block centroid. Typically we combined only the impacts of facilities within the same source category, and assessed chronic exposure and risk only for census blocks with at least one resident (i.e., locations where people may reasonably be assumed to reside rather than receptor points at the fenceline of a facility). Chronic ambient concentrations were calculated as the annual average of all estimated short-term (one-hour) concentrations at each block centroid. Possible future residential use of currently uninhabited areas was not

considered. Census blocks, the finest resolution available in the census data, are typically comprised of approximately 40 people or about ten households. For each facility in this source category census block locations were carefully evaluated for proximity to each facility's property line (see Appendix 7).

In contrast to the development of ambient concentrations for evaluating long-term exposures, which was performed only for occupied census blocks, worst-case short-term (one-hour) concentrations were estimated both at the census block centroids and at points nearer the facility that represent locations where people may be present for short periods, but generally no nearer than 100 meters from the center of the facility (note that for large facilities, this 100-meter ring could still contain locations inside the facility property). Since short-term emission rates were needed to screen for the potential for hazard via acute exposures, and since the ICR contains only annual emission totals, we generally apply the assumption to all source categories that the maximum one-hour emission rate from any source is ten times the average annual hourly emission rate for that source.

The average hourly emissions rate is defined as the total emissions for a year divided by the total number of operating hours in the year. The choice of a factor of ten for acute screening was originally based on engineering judgment. To develop a more robust peak-to-mean emissions factor, and in response to one of the key messages from the SAB consultation on our RTR Assessment Plan, we performed an analysis using a short-term emissions dataset from a number of sources located in Texas (originally reported on by Allen *et al.* 2004)[*7*]. In that report, the Texas Environmental Research Consortium Project compared hourly and annual emissions data for volatile organic compounds for all facilities in a heavily-industrialized 4-county area (Harris, Galveston, Chambers, and Brazoria Counties, TX) over an eleven-month time period in 2001. We obtained the dataset and performed our own analysis, focusing that analysis on sources which reported emitting high quantities of HAP over short periods of time (see Appendix 3, *Analysis of data on short-term emission rates relative to long-term emission rates*). Most peak emission events were less than twice the annual average, the highest was a factor of 74 times the annual average, and the 99^{th} percentile ratio of peak hourly emission rate to the annual hourly emission rate was 9. Based on these results, we typically chose a factor of ten for the initial screening. However, for the pulp and paper source category we have maximum hourly emissions estimates for each process group that indicate that a factor of 2 is more appropriate for this source category. These factors are intended to cover all possible hourly peaks associated with routinely-variable emissions. While there have been some documented emission excursions above this level, our analysis of the data from the Texas Environmental Research Consortium suggests that this factor should cover more than 99 percent of the short-term peak gaseous or volatile HAP emissions from typical industrial sources. We have no data relating specifically to peak short-term emissions of particulate HAP. In the absence of source category-specific data, we use this same default approach for particulate emissions as well.

Census block elevations for HEM3 modeling were determined nationally from the US Geological Service 1-degree digital elevation model (DEM) data files, which have a spatial resolution of about 90 meters. Elevations of polar grid points used in estimating short- and long-term ambient concentrations were assumed to be equal to the highest elevation of any

census block falling within the polar grid sector corresponding to the grid point. If a sector does not contain any blocks, the model defaults the elevation to that of the nearest block. If an elevation is not provided for the emission source, the model uses the average elevation of all sectors within the innermost model ring.

In addition to using receptor elevation to determine plume height, AERMOD adjusts the plume's flow if nearby elevated hills are expected to influence the wind patterns. For details on how hill heights were estimated and used in the AERMOD modeling see Appendix 1.

2.3 Estimating human inhalation exposure

We used the estimated annual average ambient air concentration of each HAP at each census block centroid as a surrogate for the lifetime inhalation exposure concentration of all the people who reside in the census block. That is, the risk analysis did not consider either the short-term or long-term behavior (mobility) of the exposed populations and its potential influence on their exposure.

We did not address short-term human activity for two reasons. First, our experience with the NATA assessments (which modeled daily activity using EPA's HAPEM model) suggests that, given our current understanding of microenvironment concentrations and daily activities, modeling short-term activity would, on average, reduce risk estimates about 25 percent for particulate HAP; it will also reduce risk estimates for gaseous HAP, but typically by much less. Second, basing exposure estimates on average ambient concentrations at census block centroids may underestimate or overestimate actual exposure concentrations at some residences. Further reducing exposure estimates for the most highly exposed residents by modeling their short-term behavior could add a systematic low bias to these results.

We did not address long-term migration nor population growth or decrease over 70 years, instead basing the assessment on the assumption that each person's predicted exposure is constant over the course of their lifetime which is assumed to be 70 years. In assessing cancer risk, we generally estimated three metrics; the maximum individual risk (MIR), which is defined as the risk associated with a lifetime of exposure at the highest concentration; the population risk distribution; and the cancer incidence. The assumption of not considering short or long-term population mobility does not bias the estimate of the theoretical MIR nor does it affect the estimate of cancer incidence since the total population number remains the same. It does, however, affect the shape of the distribution of individual risks across the affected population, shifting it toward higher estimated individual risks at the upper end and reducing the number of people estimated to be at lower risks, thereby increasing the estimated number of people at specific risk levels.

When screening for potentially significant acute exposures, we used an estimate of the highest hourly ambient concentration at any off-site location as the surrogate for the maximum potential acute exposure concentration for any individual.

2.4 Acute Risk Screening and Refined Assessments

In establishing a scientifically defensible approach for the assessment of potential health risks due to acute exposures to HAP, we followed the same general approach that has been used for

developing chronic health risk assessments under the residual risk program. That is, we developed a tiered, iterative approach. This approach to risk assessment was endorsed by the National Academy of Sciences in its 1993 publication "Science and Judgment in Risk Assessment" and subsequently was adopted in the EPA's "Residual Risk Report to Congress" in 1999.

The assessment methodology is designed to eliminate from further consideration those facilities for which we have confidence that no acute adverse health effects of concern will occur. To do so, we use what is called a tiered, iterative approach to the assessment. This means that we begin with a screening assessment, which relies on readily available data and uses conservative assumptions that in combination approximate a worst-case exposure. The result of this screening process is that either the facility being assessed poses no potential acute health risks (i.e., it "screens out"), or that it requires further, more refined assessment. A refined assessment could use industry- or site-specific data on the temporal pattern of emissions, the layout of emission points at the facility, the boundaries of the facility, and/or the local meteorology. In some cases, all of these site-specific data would be needed to refine the assessment; in others, lesser amounts of site-specific data can be used to determine that acute exposures are not a concern, and significant additional data collection is not necessary.

Acute health risk screening was performed for each facility as the first step. We used conservative assumptions for emission rates, meteorology, and exposure location. We used the following worst-case assumptions in our screening approach:

- Peak 1-hour emissions were assumed to equal 10 times the average 1-hour emission rates.
- For facilities with multiple emission points, peak 1-hour emissions were assumed to occur at all emission points at the same time.
- For facilities with multiple emission points, 1-hour concentrations at each receptor were assumed to be the sum of the maximum concentrations due to each emission point, regardless of whether those maximum concentrations occurred during the same hour.
- Worst-case meteorology (from one year of local meteorology) was assumed to occur at the same time the peak emission rates occur. The recommended EPA local-scale dispersion model, AERMOD, is used for simulating atmospheric dispersion.
- A person was assumed to be located downwind at the point of maximum impact during this same worst-case 1-hour period, but no nearer to the source than 100 meters.
- The maximum impact was compared to multiple short-term health benchmarks for the HAP being assessed to determine if a possible acute health risk might exist. These benchmarks are described in section 2.6 of this report.

As mentioned above, when we identify acute impacts which exceed their relevant benchmarks, we pursue refining our acute screening estimates to the extent possible. In some cases, this includes the use of a refined emissions multiplier to estimate the peak hourly emission rates from the average rates (rather than the default factor of 10). In other cases, this entails determining the actual physical layout and boundaries of a facility to more accurately

gauge where people might reasonable be exposed for an hour. For the pulp and paper source category, maximum hourly emissions estimates were available by emission process group, so we did not use the default emissions multiplier of 10. The memorandum entitled, *Inputs to the Pulp and Paper Industry October 2011 Residual Risk Modeling* includes a detailed description of how the maximum hourly emissions were developed for this source category and can be found in the docket for this rule making. We also conducted a review of the layout of emission points at the facilities with the facility boundaries to determine the maximum off-site acute impact for the facilities that did not screen out during the initial model run. Refer to Appendices 5 and 6 for the detailed results for these sites.

2.5 Multipathway and environmental risk screening

The potential for significant human health risks due to exposures via routes other than inhalation (i.e., multipathway exposures) was screened by first determining whether any sources emitted any hazardous air pollutants known to be persistent and bioaccumulative in the environment (PB-HAP)[1]. The PB-HAP compounds or compound classes are identified for the screening from the EPA's Air Toxics Risk Assessment Library [*8*]. Examples of PB-HAP are cadmium compounds, chlordane, chlorinated dibenzodioxins and furans, DDE, heptachlor, hexachlorobenzene, hexachlorocyclohexane, lead compounds, mercury compounds, methoxychlor, polychlorinated biphenyls, polycyclic organic matter (POM), toxaphene, and trifluralin. Emissions of cadmium, lead, mercury, and POM were identified in the emissions inventories for the pulp and paper source category.

With respect to PB-HAP emissions other than lead, emissions were evaluated for potential non-inhalation risks and adverse environmental impacts using our screening scenario which was developed for use with the TRIM.FaTE[2] model. This screening scenario uses environmental media outputs from the peer-reviewed TRIM.FaTE to estimate the maximum potential ingestion risks for any specified emission scenario by using a generic farming/fishing exposure scenario that simulates a subsistence environment. The screening scenario retains many of the ingestion and scenario inputs developed for EPA's Human Health Risk Assessment Protocols (HHRAP) for hazardous waste combustion facilities.[3] In the development of the screening scenario a sensitivity analysis was conducted to ensure that its key design parameters were established such that environmental media concentrations were not underestimated, and to also minimize the occurrence of false positives for human health endpoints. See Appendix 4 for a complete discussion of the development and testing of the screening scenario, as well as for the values of facility-level emission rates developed for screening potentially significant multi-pathway impacts. For the purpose of developing emission rates for our multi-pathway screening, we derived emission levels for each PB-HAP (other than lead) at which the maximum human health risk would be 1 in a million for lifetime cancer risk or a hazard quotient of 1.0 for noncancer impacts.

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[1] Although the two-letter chemical symbol for lead is Pb, in this assessment PB-HAP refers to the many air pollutants known to be persistent and bioaccumulative in the environment. In instances where the report is specifically referring to lead, it is spelled out (i.e., the two-letter chemical symbol for lead is not used in this document).

[2] EPA's Total Risk Integrated Methodology (General Information) http://epa.gov/ttn/fera/trim_gen.html

[3] EPA's Human Health Risk Assessment Protocol (HHRAP) for Hazardous Waste Combustion Facilities; http://www.epa.gov/epaoswer/hazwaste/combust/riskvol.htm#volume1

In evaluating the potential multi-pathway risks from emissions of lead compounds, rather than developing a screening emission rate for them, we compared maximum estimated chronic atmospheric concentrations with the current National Ambient Air Quality Standard (NAAQS) for lead. Values below the NAAQS were considered to have a low potential for multi-pathway risks.

The NAAQS value, a public health policy judgment, incorporated the Agency's most recent health evaluation of air effects of lead exposure for the purposes of setting a national ambient air quality standard. In setting this value, the Administrator promulgated a standard that was requisite to protect public health with an adequate margin of safety. We consider values below the level of the primary NAAQS to protect against multipathway risks because as mentioned above, the primary NAAQS is set as to protect public health with an adequate margin of safety. However, ambient air lead concentrations above the NAAQS are considered to pose the potential for increased risk to public health. We consider this NAAQS assessment to be a refined analysis given: 1) the numerous health studies, detailed risk and exposure analyses, and level of external peer and public review that went into the development of the primary NAAQS for lead, combined with: 2) the site-specific dispersion modeling used in this assessment to estimated ambient lead concentrations due to ferroalloys emissions. It should be noted, however, that this comparison does not account for possible population exposures to lead from sources other than the one being modeled; for example, via consumption of water from untreated local sources or ingestion of locally grown food. Nevertheless, the Administrator judged that such a standard would protect, with an adequate margin of safety, the health of children and other at-risk populations against an array of adverse health effects, most notably including neurological effects, particularly neurobehavioral and neurocognitive effects, in children (73 FR 67007). The Administrator, in setting the standard, also recognized that no evidence-or risk based bright line indicated a single appropriate level. Instead a collection of scientific evidence and other information was used to select the standard from a range of reasonable values (73 FR 67006).

We further note that comparing ambient lead concentrations to the NAAQS for lead, considering the level, averaging time, form and indicator, also informs whether there is the potential for adverse environmental effects. This is because the secondary lead NAAQS, set to protect against adverse welfare effects (including adverse environmental effects), has the same averaging time, form, and level as the primary standard. Thus, ambient lead concentrations above the NAAQS for lead also indicate the potential for adverse environmental effects.

Additionally, we evaluated the potential for significant ecological exposures to non PB-HAP from exceedances of chronic human health inhalation thresholds in the ambient air near these facilities. Human health dose-response threshold values are generally derived from studies conducted on laboratory animals (such as rodents) and developed with the inclusions of uncertainty factors that could be up to 3000. As a result, these human threshold values are often significantly lower than the level expected to cause an adverse effect in an exposed rodent. As such, we have concluded that terrestrial mammalian receptors are unlikely to be at risk of adverse effects due to inhalation exposures from non PB-HAP. For this source

category, EPA considered effects to the environment separate from human health risk in order to determine whether it is necessary to set a more stringent standard to prevent an adverse environmental effect. In considering effects to the environment, EPA first determined that some HAPs of potential concern with respect to the environment are emitted from sources in this category. These HAPs are hydrogen chloride, chlorine, POM, mercury, and cadmium. The agency also determined that there was at least some potential for exposures to environmental receptors, because the presence of such receptors around the sources in this category cannot be ruled out. EPA then determined that given the small amount of HAP that is emitted from sources in this category, the agency does not expect an environment effect to occur.

2.6 Dose-Response Assessment

2.6.1 Sources of chronic dose-response information

Dose-response assessment (carcinogenic and non-carcinogenic) for chronic exposure (either by inhalation or ingestion) for the HAPs reported in the emissions inventory for the pulp and paper source category were based on the EPA Office of Air Quality Planning and Standards' existing recommendations for HAPs [*9*], also used for NATA [*10*]. This information has been obtained from various sources and prioritized according to (1) conceptual consistency with EPA risk assessment guidelines and (2) level of peer review received. The prioritization process was aimed at incorporating into our assessments the best available science with respect to dose-response information. The recommendations are based on the following sources, in order of priority:

1) US Environmental Protection Agency (EPA). EPA has developed dose-response assessments for chronic exposure for many of the pollutants in this study. These assessments typically provide a qualitative statement regarding the strength of scientific data and specify a reference concentration (RfC, for inhalation) or reference dose (RfD, for ingestion) to protect against effects other than cancer and/or a unit risk estimate (URE, for inhalation) or slope factor (SF, for ingestion) to estimate the probability of developing cancer. The RfC is defined as an "estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime." The RfD is "an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime." The URE is defined as "the upper-bound excess cancer risk estimated to result from continuous lifetime exposure to an agent at a concentration of 1 $\mu g/m^3$ in air." The SF is "an upper bound, approximating a 95 percent confidence limit, on the increased cancer risk from a lifetime exposure to an agent. This estimate, [is] usually expressed in units of proportion (of a population) affected per mg/kg-day…" EPA disseminates dose-response assessment information in several forms, based on the level of review. The Integrated Risk Information System (IRIS) [*11*] is an EPA database that contains scientific health assessment information, including dose-response information. All IRIS assessments since 1996 have also undergone independent external peer review. The current IRIS process includes review by EPA scientists, interagency reviewers from other federal

agencies, and the public, and peer review by independent scientists external to EPA. New IRIS values are developed and old IRIS values are updated as new health effects data become available. Refer to the "IRIS Track" website for detailed information on status and scheduling of current individual IRIS assessments and updates (http://cfpub.epa.gov/ncea/iristrac/index.cfm). EPA's science policy approach, under the current carcinogen guidelines, is to use linear low-dose extrapolation as a default option for carcinogens for which the mode of action (MOA) has not been identified. We expect future EPA dose-response assessments to identify nonlinear MOAs where appropriate, and we will use those analyses (once they are peer reviewed) in our risk assessments. At this time, however, there are no available carcinogen dose-response assessments for inhalation exposure that are based on a nonlinear MOA.

2) US Agency for Toxic Substances and Disease Registry (ATSDR). ATSDR, which is part of the US Department of Health and Human Services, develops and publishes Minimum Risk Levels (MRLs) [*12*] for inhalation and oral exposure to many toxic substances. As stated on the ATSDR web site: "Following discussions with scientists within the Department of Health and Human Services (HHS) and the EPA, ATSDR chose to adopt a practice similar to that of the EPA's Reference Dose (RfD) and Reference Concentration (RfC) for deriving substance specific health guidance levels for non neoplastic endpoints." The MRL is defined as "an estimate of daily human exposure to a substance that is likely to be without an appreciable risk of adverse effects (other than cancer) over a specified duration of exposure." ATSDR describes MRLs as substance-specific estimates to be used by health assessors to select environmental contaminants for further evaluation. Exposures above an MRL do not necessarily represent a threat, and MRLs are therefore not intended for use as predictors of adverse health effects or for setting cleanup levels.

3) California Environmental Protection Agency (CalEPA). The CalEPA Office of Environmental Health Hazard Assessment has developed dose-response assessments for many substances, based both on carcinogenicity and health effects other than cancer. The process for developing these assessments is similar to that used by EPA to develop IRIS values and incorporates significant external scientific peer review. As cited in the CalEPA Technical Support Document for developing their chronic assessments[4]: "The guidelines for developing chronic inhalation exposure levels incorporate many recommendations of the U.S. EPA [*13*] and NAS [*14*]." The non-cancer information includes available inhalation health risk guidance values expressed as chronic inhalation reference exposure levels (RELs) [*15*]. CalEPA defines the REL as "the concentration level at or below which no health effects are anticipated in the general human population." CalEPA's quantitative dose-response information on carcinogenicity by inhalation exposure is expressed in terms of the URE [*16*], defined similarly to EPA's URE.

In developing chronic risk estimates, we adjusted dose-response values for some HAPs based on professional judgment, as follows:

[4] Air Toxics Hot Spots Program, Risk Assessment Guidelines, Part III - Technical Support Document for the Determination of Non-cancer Chronic Reference Exposure Levels. Air Toxicology and Epidemiology Section, Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. February 2000 (http://www.oehha.ca.gov/air/chronic_rels/pdf/relsP32k.pdf)

1) In the case of HAP categories such as glycol ethers and cyanide compounds, the most conservative dose-response value of the chemical category is used as a surrogate for other compounds in the group for which dose-response values are not available. This is done in order to examine, under conservative assumptions, whether these HAPs that lack dose-response values may pose an unacceptable risk and require further examination, or screen out from further assessment.

2) Where possible for emissions of unspecified mixtures of HAP categories such as metal compounds and POM, we apply category-specific chemical speciation profiles appropriate to the source category to develop a composite dose-response value for the category.

3) In 2004, the EPA determined that the Chemical Industry Institute of Toxicology (CIIT) cancer dose-response value for formaldehyde (5.5×10^{-9} per $\mu g/m^3$) was based on better science than the IRIS cancer dose-response value (1.3×10^{-5} per $\mu g/m^3$), and we switched from using the IRIS value to the CIIT value in risk assessments supporting regulatory actions. Subsequent research published by EPA suggested that the CIIT model was not appropriate and in 2010 EPA returned to using 1991 IRIS value. EPA has been working on revising the formaldehyde IRIS assessment and the National Academy of Sciences (NAS) completed its review of the EPA's draft assessment in April of 2011.[5] EPA will follow the NAS Report recommendations and will present results obtained by implementing the biologically-based dose-response (BBDR) model for formaldehyde. EPA will compare these estimates with those currently presented in the External Review draft of the assessment and will discuss their strengths and weaknesses. As recommended by the NAS committee, appropriated sensitivity and uncertainty analyses will be an integral component of implementing the BBDR model. In the interim, we will present findings using the 1991 IRIS value as a primary estimate and EPA may also consider other information as the science evolves.

4) In the case of nickel compounds, to provide a health-protective estimate of potential cancer risks, we used the IRIS URE value for nickel subsulfide in this assessment. Based on past scientific and technical considerations, the determination of the percent of nickel subsulfide was considered a major factor for estimating the extent and magnitude of the risks of cancer due to nickel-containing emissions. Nickel speciation information for some of the largest nickel-emitting sources (including oil combustion, coal combustion, and others) suggested that at least 35 percent of total nickel emissions may be soluble compounds and that the URE for the mixture of inhaled nickel compounds (based on nickel subsulfide, and representative of pure insoluble crystalline nickel) could be derived to reflect the assumption that 65 percent of the total mass of nickel may be carcinogenic. Based on consistent views of major scientific bodies (i.e., National Toxicology Program (NTP) in their 12th Report of the Carcinogens (ROC)[6], International Agency for Research

[5] http://www.nap.edu/catalog.php?record_id=13142

[6] National Toxicology Program (NTP), 2011. Report on carcinogens. 12th ed. Research Triangle Park, NC: US Department of Health and Human Services (DHHS), Public Health Service. Available online at http://ntp.niehs.nih.gov/ntp/roc/twelfth/roc12.pdf

on Cancer (IARC)[7], and other international agencies)[8] that consider all nickel compounds to be carcinogenic, we currently consider all nickel compounds to have the potential of being as carcinogenic to humans. The *12th Report of the Carcinogens* states that the "combined results of epidemiological studies, mechanistic studies, and carcinogenic studies in rodents support the concept that nickel compounds generate nickel ions in target cells at sites critical for carcinogenesis, thus allowing consideration and evaluation of these compounds as a single group." Although the precise nickel compound (or compounds) responsible for the carcinogenic effects in humans is not always clear, studies indicate that nickel sulfate and the combinations of nickel sulfides and oxides encountered in the nickel refining industries cause cancer in humans (these studies are summarized in a review by Grimsrud et al., 2010[9]). The major scientific bodies mentioned above have also recognized that there are differences in toxicity and/or carcinogenic potential across the different nickel compounds. In this inhalation risk assessment, to take a conservative approach, we have considered all nickel compounds to be as carcinogenic as nickel subsulfide and have applied the IRIS URE for nickel subsulfide without a factor to reflect the assumption that 100 percent of the total mass of nickel may be as carcinogenic as pure nickel subsulfide. In addition, given that there are two additional URE[10] values derived for exposure to mixtures of nickel compounds, as a group, that are 2-3 fold lower than the IRIS URE for nickel subsulfide, the EPA also considers it reasonable to use a value that is 50 percent of the IRIS URE for nickel subsulfide for providing an estimate of the lower end of the plausible range of cancer potency values for different mixtures of nickel compounds.

5) A substantial proportion of POM reported to EPA's National Emission Inventory (NEI) is not speciated into individual compounds. As a result, it is necessary to apply the same simplifying assumptions to assessments that are used in NATA [*17*]. The NATA approach partitions POM into eight different non-overlapping "groups" that are modeled as separate pollutants. Each POM group comprises POM species of similar carcinogenic potency, for which we can apply the same URE.

6) A chronic screening level of 163 ug/m^3 was developed for carbonyl sulfide (COS) from a No Observed Adverse Effects Level (NOAEL) of 200 ppm based on brain lesions and neurophysiological alterations in rodents. A more detailed discussion of the studies used to develop the COS chronic screening level is provided in Appendix 8. The screening level includes a total uncertainty factor (UF) of 3,000: 10x for extrapolation for

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[7] International Agency for Research on Cancer (IARC), 1990. IARC monographs on the evaluation of carcinogenic risks to humans. Chromium, nickel, and welding. Vol. 49. Lyons, France: International Agency for Research on Cancer, World Health Organization Vol. 49:256.

[8] World Health Organization (WHO, 1991) and the European Union's Scientific Committee on Health and Environmental Risks (SCHER, 2006).

[9] Grimsrud TK and Andersen A. Evidence of carcinogenicity in humans of water-soluble nickel salts. J Occup Med Toxicol 2010, 5:1-7. Available online at http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2868037/?tool=pubmed.

[10] Two UREs (other than the current IRIS values) have been derived for nickel compounds as a group: one developed by the California Department of Health Services (http://www.arb.ca.gov/toxics/id/summary/nickel_tech_b.pdf) and the other by the Texas Commission on Environmental Quality (http://www.epa.gov/ttn/atw/nata1999/99pdfs/healtheffectsinfo.pdf).

interspecies differences, 10x for consideration of intraspecies variability, 10x for extrapolation from subchronic to chronic duration, and 3x for database insufficiencies. See section 5 of this document for a detailed discussion of exposure modeling uncertainties. The chronic screening level for COS is used only as a screening level assessment to identify areas with significant inhalation risk potential. A high COS chronic risk based on the screening level does not necessarily indicate that further action is required.

7) For 1 carcinogenic substance, (propylene dichloride) that lack inhalation assessments from the sources evaluated in this document, oral carcinogenic potency estimates were converted to inhalation UREs. The conversion from oral risk (per mg/kg/d oral intake) to inhalation risk (per $\mu g/m^3$ inhaled) was based on EPA's standard assumptions of a 70-kg body mass and 20 m^3/d inhalation rate, as follows:

$$URE\left(\frac{\mu g}{m^3}\right)^{-1} = CPS\left(\frac{mg}{kg \cdot d}\right)^{-1} \times \frac{1}{70\,kg} \times 20\left(\frac{m^3}{d}\right) \times \frac{1}{1000}\left(\frac{mg}{\mu g}\right)$$

Where: URE = Unit risk estimate for inhalation (risk per $\mu g/m^3$)
CPS = Carcinogenic potency slope for ingestion (risk per mg oral intake per kg body mass per day)

EPA understands that conversion of oral dose-response information to inhalation exposure may add significant uncertainty to the resulting risk estimates. However, the alternative to this would have been to omit these substances from quantitative inhalation risk estimates altogether, thereby making a *de facto* assumption of zero carcinogenic potency. For the purposes of the residual risk assessment, EPA prefers to use the approach described above to screen these carcinogens for their potential contributions to risk. If a substance is determined to be a potentially important contributor to risk, it will be prioritized for further dose-response development through EPA's IRIS process.

The emissions inventory for the pulp and paper source category includes emissions of HAP with available chronic quantitative inhalation dose-response values. Of these, 47 are classified as known, probable, or possible carcinogens, with quantitative cancer dose-response values available. These 47 HAP, their quantitative inhalation chronic cancer dose-response values, and the source of each value are listed in Table 2.6-1(a). The POM compounds with chronic oral cancer dose-response values available (for which multipathway screening assessments were performed) are listed in Table 2.6-1(b). Seventy-seven HAP have quantitative inhalation chronic noncancer threshold values available, two of these HAP (mercury and cadmium), for which a multipathway assessment was performed, also have quantitative oral chronic noncancer threshold values available. These 77 HAP, their threshold values, and the source of the value are listed in Table 2.6-2(a) and Table 2.6-2(b).

Table 2.6-1(a) Dose-Response Values for Chronic Inhalation Exposure to Carcinogens

URE (unit risk estimate for cancer)[11] = cancer risk per μg/m^3 of average lifetime exposure. Sources: IRIS = EPA Integrated Risk Information System, EPA ORD = EPA Office of Research & Development, EPA OAQPS = EPA Office of Air Quality Planning & Standards, CAL = California EPA Office of Environmental Health Hazard Assessment. HEAST = EPA Health Effects Assessment Tables, Conv. Oral = Oral unit risk converted to inhalation.

Pollutant	CAS Number[12]	URE[11] (1/μg/m3)	Source
1,1,2,2-Tetrachloroethane	79345	0.000058	IRIS
1,1,2-Trichloroethane	79005	0.000016	IRIS
1,3-Butadiene	106990	0.00003	IRIS
1,4-Dichlorobenzene	106467	0.000011	CAL
2,4,6-Trichlorophenol	88062	0.0000031	IRIS
2,4-Toluene diamine	95807	0.0011	CAL
2-Nitropropane	79469	0.0000056	EPA OAQPS
Acetaldehyde	75070	0.0000022	IRIS
Acrylamide	79061	0.00016	IRIS
Acrylonitrile	107131	0.000068	IRIS
Aniline	62533	0.0000016	CAL
Arsenic compounds	7440382	0.0043	IRIS
Benzene[13]	71432	0.0000078	IRIS
Beryllium compounds	7440417	0.0024	IRIS
Bis(2-ethylhexyl)phthalate	117817	0.0000024	CAL
Bromoform	75252	0.0000011	IRIS
Cadmium compounds	7440439	0.0018	IRIS
Carbon tetrachloride	56235	0.000006	IRIS
Chlorobenzilate	510156	0.000078	HEAST
Chromium (VI) compounds	18540299	0.012	IRIS
Ethyl benzene	100414	0.0000025	CAL
Ethylene dibromide	106934	0.0006	IRIS
Ethylene dichloride	107062	0.000026	IRIS
Ethylene oxide	75218	0.000088	CAL
Ethylidene dichloride (1,1-Dichloroethane)	75343	0.0000016	CAL
Formaldehyde[14]	50000	0.000013	IRIS
Hexachloroethane	67721	0.000004	IRIS

[11] The URE is the upper-bound excess cancer risk estimated to result from continuous lifetime exposure to an agent at a concentration of 1 μg/m^3 in air. URE's are considered upper bound estimates meaning they represent a plausible upper limit to the true value.

[12] Chemical Abstract Services identification number. For groups of compounds that lack a CAS number we have used a surrogate 3-digit identifier corresponding to the group's position on the CAA list of HAPs.

[13] The EPA IRIS assessment for benzene provides a range of plausible UREs. This assessment used the highest value in that range, 7.8E-06 per ug/m^3. The low end of the range is 2.2E-06 per ug/m^3.

[14] The EPA has used the CIIT URE value, 5.5X10^{-9} per mg/m^3, to characterize formaldehyde cancer risk in some instances.

Table 2.6-1(a) Dose-Response Values for Chronic Inhalation Exposure to Carcinogens

URE (unit risk estimate for cancer)[11] = cancer risk per µg/m^3 of average lifetime exposure. Sources: IRIS = EPA Integrated Risk Information System, EPA ORD = EPA Office of Research & Development, EPA OAQPS = EPA Office of Air Quality Planning & Standards, CAL = California EPA Office of Environmental Health Hazard Assessment. HEAST = EPA Health Effects Assessment Tables, Conv. Oral = Oral unit risk converted to inhalation.

Pollutant	CAS Number[12]	URE[11] (1/µg/m3)	Source
Methylene chloride	75092	0.00000047	IRIS
Naphthalene	91203	0.000034	CAL
Nickel compounds	7440020	0.00048	EPA OAQPS[15]
Nitrobenzene	98953	0.00004	IRIS
Pentachlorophenol	87865	0.0000051	CAL
Polycyclic Organic Matter[16] (POM)			
7,12-Dimethylbenz(a)anthracene	57976	0.1136	CAL
3-Methylcholanthrene	56495	0.01008	CAL
Benz(a)anthracene	56553	0.000176	CAL
Benzo(a)pyrene	50328	0.00176	CAL
Benzo(b)fluoranthene	205992	0.000176	CAL
Benzo(k)fluoranthene	207089	0.000176	CAL
Chrysene	218019	0.0000176	CAL
Dibenzo(a,h)anthracene	53703	0.0019184	CAL
Indeno(1,2,3-c,d)pyrene	193395	0.000176	CAL
POM 71002	187	0.000088	CAL
POM 72002	187	0.000088	CAL
Propylene dichloride[17]	78875	0.000019	Conv. Oral
Tetrachloroethene	127184	0.0000059	CAL
Trichloroethylene	79016	0.000002	CAL
Vinyl chloride	75014	0.0000088	IRIS

[15] The EPA IRIS assessments for nickel compounds provide a range of plausible UREs. This assessment used the highest value in that range which is equal to the URE for nickel subsulfide, 4.8E-04 per ug/m^3. The low end of the range is equal to 50% of the URE for nickel subsulfide, 2.4E-04 per ug/m^3.

[16] POM without a chemical-specific URE are assigned a URE associated with a mixture of POM compounds having similar characteristics. Details of this method, also used in the 2002 National Air Toxics Assessment, are available at http://www.epa.gov/ttn/atw/nata2002/02pdfs/pom_approach.pdf.

[17] No inhalation unit risk estimates were available for this compound. Therefore we converted from a oral potency slope of 0.068 per mg/kg/d. UREs that are converted from the oral route to the inhalation route of exposure are considered highly uncertain, and are only used in cases where no other URE is available.

Table 2.6-1(b) Dose-Response Values for Chronic Oral Exposure to Carcinogens

SF (oral slope factor for cancer) = cancer risk per mg/kg/d of average lifetime exposure. Sources: IRIS = EPA Integrated Risk Information System, CAL = California EPA Office of Environmental Health Hazard Assessment, EPA/OAQPS = interim value recommended by the EPA Office of Air Quality Planning and Standards, EPA ORD = EPA Office of Research and Development, HEAST = EPA Health Effects Assessment Tables

Pollutant	CAS Number	SF (1/mg/kg/d)	Source
Polycyclic organic matter (POM)			
Benzo(a)anthracene	56553	1.2	CAL
Benzo(a)pyrene	50328	7.3	IRIS
Benzo(b)fluoranthene	205992	1.2	CAL
Benzo(k)fluoranthene	207089	1.2	CAL
Chrysene	218019	0.12	CAL
Dibenz(a,h)anthracene	53703	4.1	CAL
7,12-Dimethylbenz(a)anthracene	57976	250	CAL
Indeno(1,2,3-cd)pyrene	193395	1.2	CAL
3-Methlycholanthrene	56495	22	CAL

Table 2.6-2(a) Dose-Response Values for Chronic Inhalation Exposure to Noncarcinogens

RfC (reference inhalation concentration) = an estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Sources: IRIS = EPA Integrated Risk Information System, CAL = California EPA Office of Environmental Human Health Assessment, ATSDR = US Agency for Toxic Substances Disease Registry, HEAST = EPA Health Effects Assessment Tables, EPA OAQPS = EPA Office of Air Quality Planning & Standards, EPA ORD = EPA Office of Research and Development

Pollutant	CAS Number[12]	RfC (mg/m^3)	Source[18]
1,1,1-Trichloroethane (methyl chloroform)	71556	5	IRIS - M
1,1,2-Trichloroethane	79005	0.4	CAL
1,2,4-Trichlorobenzene	120821	0.2	HEAST
1,3-Butadiene	106990	0.002	IRIS - M
1,4-Dichlorobenzene	106467	0.8	IRIS - M
2-Nitropropane	79469	0.02	IRIS - L
Acetaldehyde	75070	0.009	IRIS - L
Acetonitrile	75058	0.06	IRIS - M
Acrylamide	79061	0.006	IRIS - M
Acrylonitrile	107131	0.002	IRIS - M

[18] The descriptors L (low), M (medium), and H (high) have been added for IRIS RfC values to indicate the overall level of confidence in the RfC value, as reported in IRIS.

Table 2.6-2(a) Dose-Response Values for Chronic Inhalation Exposure to Noncarcinogens

RfC (reference inhalation concentration) = an estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Sources: IRIS = EPA Integrated Risk Information System, CAL = California EPA Office of Environmental Human Health Assessment, ATSDR = US Agency for Toxic Substances Disease Registry, HEAST = EPA Health Effects Assessment Tables, EPA OAQPS = EPA Office of Air Quality Planning & Standards, EPA ORD = EPA Office of Research and Development

Pollutant	CAS Number[12]	RfC (mg/m^3)	Source[18]
Aniline	62533	0.001	IRIS - L
Antimony compounds	7440360	0.0002	IRIS - L
Arsenic compounds	7440382	0.000015	CAL
Benzene	71432	0.03	IRIS - M
Beryllium compounds	7440417	0.00002	IRIS - M
Bis(2-ethylhexyl)phthalate	117817	0.01	CAL
Cadmium compounds	7440439	0.00001	ATSDR
Carbon disulfide	75150	0.7	IRIS - M
Carbon tetrachloride	56235	0.1	IRIS - M
Carbonyl sulfide	463581	0.163	EPA ORD[19]
Chlorine	7782505	0.00015	ATSDR
Chlorobenzene	108907	1	CAL
Chloroform	67663	0.098	ATSDR
Chromium (VI) compounds	18540299	0.0001	IRIS - M
Cobalt compounds	7440484	0.0001	ATSDR
Cresols (mixed)	1319773	0.6	CAL
m-Cresol	108394	0.6	CAL
o-Cresol	95487	0.6	CAL
p-Cresol	106445	0.6	CAL
Cumene	98828	0.4	IRIS - M
Cyanide & Cyanide Compounds[20]			
Cyanide compounds	57125	0.0008	IRIS – L/M
Hydrogen cyanide	74908	0.0008	IRIS – L/M
Diethanolamine	111422	0.003	CAL
Ethyl benzene	100414	1	IRIS - L
Ethylene dibromide	106934	0.009	IRIS - M
Ethylene dichloride	107062	2.4	ATSDR
Ethylene glycol	107211	0.4	CAL
Ethylene oxide	75218	0.03	CAL
Ethylidene dichloride (1,1-Dichloroethane)	75343	0.5	HEAST
Formaldehyde	50000	0.0098	ATSDR

[19] A chronic screening level of 0.163 mg/m^3 was developed for carbonyl sulfide by EPA ORD from a No Observed Adverse Effects Level of 200 ppm based on brain lesions and neurophysiological alteration in rodents.
[20] The value for hydrogen cyanide was used as a surrogate for all cyanide compounds without an RfC.

Table 2.6-2(a) Dose-Response Values for Chronic Inhalation Exposure to Noncarcinogens

RfC (reference inhalation concentration) = an estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Sources: IRIS = EPA Integrated Risk Information System, CAL = California EPA Office of Environmental Human Health Assessment, ATSDR = US Agency for Toxic Substances Disease Registry, HEAST = EPA Health Effects Assessment Tables, EPA OAQPS = EPA Office of Air Quality Planning & Standards, EPA ORD = EPA Office of Research and Development

Pollutant	CAS Number[12]	RfC (mg/m^3)	Source[18]
Glycol Ethers[21]			
1,2-Dimethoxyethane	110714	0.02	IRIS - M
Butyl carbitol acetate	124174	0.02	IRIS - M
Ethylene glycol ethyl ether	110805	0.2	IRIS - M
Methyl cellosolve acrylate	3121617	0.02	IRIS - M
Hexachlorocyclopentadiene	77474	0.0002	IRIS – M/H
Hexachloroethane	67721	0.08	CAL
Hexane	110543	0.7	IRIS - M
Hydrochloric acid (hydrogen chloride)	7647010	0.02	IRIS - L
Lead compounds	7439921	0.00015	EPA OAQPS
Manganese compounds	7439965	0.00005	IRIS - M
Mercury and Mercury Compounds			
Mercuric chloride	7487947	0.0003	IRIS - M
Mercury (elemental)	7439976	0.0003	IRIS - M
Methanol	67561	4	CAL
Methyl bromide	74839	0.005	IRIS - H
Methyl chloride	74873	0.09	IRIS - M
Methyl isobutyl ketone	108101	3	IRIS - L/M
Methylene chloride	75092	1	ATSDR
Naphthalene	91203	0.003	IRIS - M
Nickel compounds	7440020	0.00009	ATSDR
Nitrobenzene	98953	0.009	IRIS - M
Pentachlorophenol	87865	0.1	CAL
Phenol	108952	0.2	CAL
Propionaldehyde	123386	0.008	IRIS – L/M
Propylene dichloride	78875	0.004	IRIS - M
Selenium compounds	7782492	0.02	CAL
Styrene	100425	1	IRIS - M
Tetrachloroethene	127184	0.27	ATSDR
Toluene	108883	5	IRIS - H
Trichloroethylene	79016	0.6	CAL
Triethylamine	121448	0.007	IRIS - L
Vinyl acetate	108054	0.2	IRIS - H

[21] The RfC value for ethylene glycol methyl ether (0.02 mg/m^3) was used as a surrogate for all glycol ethers without an RfC.

Table 2.6-2(a) Dose-Response Values for Chronic Inhalation Exposure to Noncarcinogens

RfC (reference inhalation concentration) = an estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime.
Sources: IRIS = EPA Integrated Risk Information System, CAL = California EPA Office of Environmental Human Health Assessment, ATSDR = US Agency for Toxic Substances Disease Registry, HEAST = EPA Health Effects Assessment Tables, EPA OAQPS = EPA Office of Air Quality Planning & Standards, EPA ORD = EPA Office of Research and Development

Pollutant	CAS Number[12]	RfC (mg/m^3)	Source[18]
Vinyl chloride	75014	0.1	IRIS - M
Vinylidene chloride	75354	0.2	IRIS – H/M
Xylenes (mixed)	1330207	0.1	IRIS - M
m-Xylene[22]	108383	0.1	IRIS - M
o-Xylene[22]	95476	0.1	IRIS - M
p-Xylene[22]	106423	0.1	IRIS - M

Table 2.6-2(b) Dose-Response Values for Chronic Oral Exposure to Noncarcinogens

RfD (reference dose) = an estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous oral exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime.
Sources: IRIS = EPA Integrated Risk Information System

Pollutant	CAS Number	RfD (mg/kg/d)	Source [23]
Mercuric chloride[24]	7439976	0.0003	IRIS - H
Cadmium compounds	7440439	0.0005	IRIS - H

2.6.2 Sources of acute dose-response information

Hazard identification and dose-response assessment information for preliminary acute inhalation exposure assessments are based on the existing recommendations of OAQPS for HAPs [*18*]. Depending on availability, the results from screening acute assessments are compared to both "no effects" reference levels for the general public, such as the California Reference Exposure Levels (RELs), as well as emergency response levels, such as Acute Exposure Guideline Levels (AEGLs) and Emergency Response Planning Guidelines (ERPGs), with the recognition that the ultimate interpretation of any potential risks associated with an estimated exceedance of a particular reference level depends on the definition of that

[22] The RfC for mixed xylene was used as a surrogate.
[23] The descriptors L (low), M (medium), and H (high) have been added for IRIS RfC values to indicate the overall level of confidence in the RfC value, as reported in the IRIS file.
[24] The multipathway exposure assessment for mercury included fate and transport analysis, that included separate oral exposure estimates for divalent mercury and methylmercury.

level and any limitations expressed therein. Comparisons among different available inhalation health effect reference values (both acute and chronic) for selected HAPs can be found in a newly released EPA document [*19*].

***California Acute Reference Exposure Levels (RELs*).** The California Environmental Protection Agency (CalEPA) has developed acute dose-response reference values for many substances, expressing the results as acute inhalation Reference Exposure Levels (RELs).

> The acute REL (http://www.oehha.ca.gov/air/pdf/acuterel.pdf) is defined by CalEPA as "the concentration level at or below which no adverse health effects are anticipated for a specified exposure duration. [*20*]. RELs are based on the most sensitive, relevant, adverse health effect reported in the medical and toxicological literature. RELs are designed to protect the most sensitive individuals in the population by the inclusion of margins of safety. Since margins of safety are incorporated to address data gaps and uncertainties, exceeding the REL does not automatically indicate an adverse health impact." Acute RELs are developed for 1-hour (and 8-hour) exposures. The values incorporate uncertainty factors similar to those used in deriving EPA's Inhalation Reference Concentrations (RfCs) for chronic exposures (and, in fact, California also has developed chronic RELs).

Acute Exposure Guideline Levels (AEGLs). AEGLs are developed by the National Advisory Committee (NAC) on Acute Exposure Guideline Levels (NAC/AEGL) for Hazardous Substances, and then reviewed and published by the National Research Council As described in the Committee's "Standing Operating Procedures (SOP)" (http://www.epa.gov/opptintr/aegl/pubs/sop.pdf), AEGLs "represent threshold exposure limits for the general public and are applicable to emergency exposures ranging from 10 min to 8 h." Their intended application is "for conducting risk assessments to aid in the development of emergency preparedness and prevention plans, as well as real time emergency response actions, for accidental chemical releases at fixed facilities and from transport carriers." The document states that "the primary purpose of the AEGL program and the NAC/AEGL Committee is to develop guideline levels for once-in-a-lifetime, short-term exposures to airborne concentrations of acutely toxic, high-priority chemicals." In detailing the intended application of AEGL values, the document states that, "It is anticipated that the AEGL values will be used for regulatory and nonregulatory purposes by U.S. Federal and State agencies, and possibly the international community in conjunction with chemical emergency response, planning, and prevention programs. More specifically, the AEGL values will be used for conducting various risk assessments to aid in the development of emergency preparedness and prevention plans, as well as real-time emergency response actions, for accidental chemical releases at fixed facilities and from transport carriers."

The NAC/AEGL defines AEGL-1 and AEGL-2 as:

> "AEGL-1 is the airborne concentration (expressed as ppm or mg/m^3) of a substance above which it is predicted that the general population, including susceptible individuals, could experience notable discomfort, irritation, or certain asymptomatic nonsensory effects. However, the effects are not disabling and are transient and reversible upon cessation of

exposure."

"AEGL-2 is the airborne concentration (expressed as ppm or mg/m^3) of a substance above which it is predicted that the general population, including susceptible individuals, could experience irreversible or other serious, long-lasting adverse health effects or an impaired ability to escape."

"Airborne concentrations below AEGL-1 represent exposure levels that can produce mild and progressively increasing but transient and nondisabling odor, taste, and sensory irritation or certain asymptomatic, nonsensory effects. With increasing airborne concentrations above each AEGL, there is a progressive increase in the likelihood of occurrence and the severity of effects described for each corresponding AEGL. Although the AEGL values represent threshold levels for the general public, including susceptible subpopulations, such as infants, children, the elderly, persons with asthma, and those with other illnesses, it is recognized that individuals, subject to unique or idiosyncratic responses, could experience the effects described at concentrations below the corresponding AEGL."

Emergency Response Planning Guidelines (ERPGs). The American Industrial Hygiene Association (AIHA) has developed Emergency Response Planning Guidelines (ERPGs) [*21*] for acute exposures at three different levels of severity. These guidelines represent concentrations for exposure of the general population (but not particularly sensitive persons) for up to 1 hour associated with effects expected to be mild or transient (ERPG-1), irreversible or serious (ERPG-2), and potentially life-threatening (ERPG-3).

ERPG values (http://www.aiha.org/insideaiha/guidelinedevelopment/erpg/Pages/default.aspx) are described in their supporting documentation as follows: "ERPGs are air concentration guidelines for single exposures to agents and are intended for use as tools to assess the adequacy of accident prevention and emergency response plans, including transportation emergency planning, community emergency response plans and incident prevention and mitigation."

ERPG-1 and ERPG-2 values are defined by AIHA as follows:

"ERPG-1 is the maximum airborne concentration below which it is believed that nearly all individuals could be exposed for up to 1 hour without experiencing other than mild transient adverse health effects or without perceiving a clearly defined, objectionable odor."

"ERPG-2 is the maximum airborne concentration below which it is believed that nearly all individuals could be exposed for up to 1 hour without experiencing or developing irreversible or other serious health effects or symptoms which could impair an individual's ability to take protective action."

The emissions inventory for the pulp and paper source category includes emissions of 50 HAP with relevant and available quantitative acute dose-response threshold values. These HAP, their acute threshold values, and the source of the value are listed below in Table 2.6-3.

Table 2.6-3 Dose-Response Values for Acute Exposure

Pollutant	CAS Number	AEGL-1 (1-hr) (mg/m^3)	AEGL-2 (1-hr) (mg/m^3)	ERPG-1 (mg/m^3)	ERPG-2 (mg/m^3)	REL
1,1,1-Trichloroethane (methyl chloroform)	71556	1300	3300	1900	3800	68
1,3-Butadiene	106990	1500	12000	22	440	
Acetaldehyde	75070	81	490	18	360	0.47
Acetonitrile	75058	22	540			
Acrylonitrile	107131	10	130	22	77	
Aniline	62533	30	46			
Arsenic compounds	7440382					0.0002
Benzene	71432	170	2600	160	480	1.3
Beryllium compounds	7440417				0.025	
Biphenyl	92524		61			
Carbon disulfide	75150	40	500	3.1	160	6.2
Carbon tetrachloride	56235	280	1200	130	630	1.9
Carbonyl sulfide	463581		140			
Chlorine	7782505	1.5	5.8	2.9	8.7	0.21
Chloroacetic acid	79118		26			
Chlorobenzene	108907	46	690			
Chloroform	67663		310		240	0.15
Cumene	98828	250	1500			
Hydrogen cyanide	74908	2.2	7.8		11	0.34
Ethyl benzene	100414	140	4800			
Ethylene dibromide	106934	130	180			
Ethylene dichloride	107062			200	810	
Ethylene oxide	75218		81		90	
Formaldehyde	50000	1.1	17	1.2	12	0.055
Glycol Ethers[25]						
1,2-Dimethoxyethane	110714					0.093
Butyl carbitol acetate	124174					0.093
Ethylene glycol ethyl ether	110805					0.37
Methyl cellosolve acrylate	3121617					0.093
Hexane	110543		12000			
Hydrochloric acid	7647010	2.7	33	4.5	30	2.1

[25] The acute REL for ethylene glycol methyl ether was used as a surrogate for glycol ether compounds without an acute REL.

Table 2.6-3 Dose-Response Values for Acute Exposure

Pollutant	CAS Number	AEGL-1 (1-hr) (mg/m^3)	AEGL-2 (1-hr) (mg/m^3)	ERPG-1 (mg/m^3)	ERPG-2 (mg/m^3)	REL
Mercury (elemental)	7439976		1.7		2	0.0006
Methanol	67561	690	2700	260	1300	28
Methyl bromide	74839		820		190	3.9
Methyl chloride	74873		1900		830	
Methyl iodide	74884			150	290	
Methylene chloride	75092	690	1900	1000	2600	14
Nickel compounds	7440020					0.006
Phenol	108952	58	89	38	190	5.8
Propionaldehyde	123386	110	620			
Styrene	100425	85	550	210	1100	21
Tetrachloroethene	127184	240	1600	680	1400	20
Toluene	108883	750	4500	190	1100	37
Trichloroethylene	79016	700	2400	540	2700	
Triethylamine	121448					2.8
Vinyl acetate	108054	24	630	18	260	
Vinyl chloride	75014	640	3100	1300	13000	180
Xylenes (mixed)	1330207	560	4000			22
m-Xylene[26]	108383					22
o-Xylene[26]	95476					22
p-Xylene[26]	106423					22

2.7 Risk Characterization

2.7.1 General

The final product of the risk assessment is the risk characterization, in which the information from the previous steps is integrated and an overall conclusion about risk is synthesized that is complete, informative, and useful for decision makers. In general, the nature of this risk characterization depends on the information available, the application of the risk information and the resources available. In all cases, major issues associated with determining the nature and extent of the risk are identified and discussed. Further, the EPA Administrator's March 1995 *Policy for Risk Characterization* [*22*] specifies that a risk characterization "be prepared in a manner that is clear, transparent, reasonable, and consistent with other risk characterizations of similar scope prepared across programs in the Agency." These principles of transparency and consistency have been reinforced by the *Agency's Risk Characterization Handbook* [*23*], in 2002 by the Agency's information quality guidelines [*24*], and in the

[26] The REL for mixed xylenes was used as a surrogate.

OMB/OSTP September 2007 Memorandum on Updated Principles for Risk Analysis[27], and are incorporated in these assessments.

Estimates of health risk are presented in the context of uncertainties and limitations in the data and methodology. Through our tiered, iterative analytical approach, we have attempted to reduce both uncertainty and bias to the greatest degree possible in these assessments, within the limitations of available time and resources. We provide summaries of risk metrics (including maximum individual cancer risks and noncancer hazards, as well as cancer incidence estimates) along with a discussion of the major uncertainties associated with their derivation to provide decision makers with the fullest picture of the assessment and its limitations.

For each carcinogenic HAP included in the assessment that has a potency estimate available, individual and population cancer risks were calculated by multiplying the corresponding lifetime average exposure estimate by the appropriate URE. This calculated cancer risk is defined as the upper-bound probability of developing cancer over a 70-yr period (i.e., the assumed human lifespan) at that exposure. Because UREs for most HAPs are upper-bound estimates, actual risks at a given exposure level may be lower than predicted, and could be zero.

For EPA's list of carcinogenic HAPs that act by a mutagenic mode-of-action [*25*], we applied EPA's *Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens* [*26*]. This guidance has the effect of adjusting the URE by factors of 10 (for children aged 0-1), 3 (for children aged 2-15), or 1.6 (for 70 years of exposure beginning at birth), as needed in risk assessments. In this case, this has the effect of increasing the estimated life time risks for these pollutants by a factor of 1.6. In addition, although only a small fraction of the total POM emissions may be reported as individual compounds, EPA expresses carcinogenic potency for compounds in this group in terms of benzo[a]pyrene equivalence, based on evidence that carcinogenic POM have the same mutagenic mechanism of action as does benzo[a]pyrene. For this reason, EPA implementation policy [*27*] recommends applying the *Supplemental Guidance* to all carcinogenic PAHs for which risk estimates are based on relative potency. Accordingly, we applied the Supplemental Guidance to all unspeciated POM mixtures.

Increased cancer incidence for the entire receptor population within the area of analysis was estimated by multiplying the estimated lifetime cancer risk for each census block by the number of people residing in that block, then summing the results for the entire modeled domain. This lifetime population incidence estimate was divided by 70 years to obtain an estimate of the number of cancer cases per year.

In the case of benzene, the high end of the reported cancer URE range was used in our

[27]Memorandum for the Heads of Executive Departments and Agencies - Updated Principles for Risk Analysis (September 19, 2007), From Susan E. Dudley, Administrator, Office of Information and Regulatory Affairs, Office of Management and Budget; and Sharon L. Hays, Associate Director and Deputy Director for Science, Office of Science and Technology Policy (http://www.whitehouse.gov/sites/default/files/omb/assets/omb/memoranda/fy2007/m07-24.pdf)

assessments to provide a conservative estimate of potential cancer risks. Use of the high end of the range provides risk estimates that are approximately 3.5 times higher than use of the equally plausible low end value. If the estimated benzene–associated risks exceed 1 in a million, we also evaluated the impact of using the low end of the URE range on our risk results.

Unlike linear dose-response assessments for cancer, noncancer health hazards generally are not expressed as a probability of an adverse occurrence. Instead, "risk" for noncancer effects is expressed by comparing an exposure to a reference level as a ratio. The "hazard quotient" (HQ) is the estimated exposure divided by a reference level (e.g., the RfC). For a given HAP, exposures at or below the reference level (HQ≤1) are not likely to cause adverse health effects. As exposures increase above the reference level (HQs increasingly greater than 1), the potential for adverse effects increases. For exposures predicted to be above the RfC, the risk characterization includes the degree of confidence ascribed to the RfC values for the compound(s) of concern (i.e., high, medium, or low confidence) and discusses the impact of this on possible health interpretations.

The risk characterization for chronic effects other than cancer is expressed in terms of the HQ for inhalation, calculated for each HAP at each census block centroid. As discussed above, RfCs incorporate generally conservative uncertainty factors in the face of uncertain extrapolations, such that an HQ greater than one does not necessarily suggest the onset of adverse effects. The HQ cannot be translated to a probability that adverse effects will occur, and is unlikely to be proportional to adverse health effect outcomes in a population.

Screening for potentially significant acute inhalation exposures also followed the HQ approach. We divided the maximum estimated acute exposure by each available short-term threshold value to develop an array of HQ values relative to the various acute endpoints and thresholds. In general, when none of these HQ values are greater than one, there is no potential for acute risk. In those cases where HQ values above one are seen, additional information is used to determine if there is a potential for significant acute risks.

2.7.2 Mixtures

Since most or all receptors in these assessments receive exposures to multiple pollutants rather than a single pollutant, we estimated the aggregate health risks associated with all the exposures from a particular source category combined.

To combine risks across multiple carcinogens, our assessments use the mixtures guidelines' [*28*,*29*] default assumption of additivity of effects, and combine risks by summing them using the independence formula in the mixtures guidelines.

In assessing noncancer hazard from chronic exposures, in cases where different pollutants cause adverse health effects via completely different modes of action, it may be inappropriate to aggregate HQs. In consideration of these mode-of-action differences, the mixtures guidelines support aggregating effects of different substances in specific and limited ways. To conform to these guidelines, we aggregated non-cancer HQs of HAPs that act by similar

toxic modes of action, or (where this information is absent) that affect the same target organ. This process creates, for each target organ, a target-organ-specific hazard index (TOSHI), defined as the sum of hazard quotients for individual HAPs that affect the same organ or organ system. All TOSHI calculations presented here were based exclusively on effects occurring at the "critical dose" (i.e., the lowest dose that produces adverse health effects). Although HQs associated with some pollutants have been aggregated into more than one TOSHI, this has been done only in cases where the critical dose affects more than one target organ. Because impacts on organs or systems that occur above the critical dose have not been included in the TOSHI calculations, some TOSHIs may have been underestimated. As with the HQ, the TOSHI should not be interpreted as a probability of adverse effects, or as strict delineation of "safe" and "unsafe" levels. Rather, the TOSHI is another measure of the potential for adverse health outcomes associated with pollutant exposure, and needs to be interpreted carefully by health scientists and risk managers.

Because of the conservative nature of the acute inhalation screening and the variable nature of emissions and potential exposures, acute impacts were screened on an individual pollutant basis, not using the TOSHI approach.

2.7.3 Facility-wide Risks

To help place the source category risks in context, we examined "facility-wide" risks using ICR data and modeling as described in Section 2.2. For the facilities in the pulp and paper source category, we estimated the maximum inhalation cancer and chronic noncancer risks associated with all HAP emissions sources at the facility, including emissions sources that are not part of the source categories but are located within a contiguous area and are under common control. We analyzed risks due to the inhalation of HAP for the populations residing within 50 kilometers of each facility. The results of the facility-wide assessment are summarized below in the Risk characterization section of this document. The complete results of the facility-wide assessment are provided in Appendix 5.

2.7.4 MACT-Allowable Risk

The emissions data in the data set for the pulp and paper source category are estimates of actual emissions on an annual basis. The risk results presented in the following sections are based on these actual emissions. To estimate emissions at the MACT-allowable level, a ratio of MACT-allowable to actual emissions for each source type was developed. This ratio was based on the level of control required by the MACT standard compared to the level of reported actual emissions and available information from the ICR on the level of control achieved by the emissions controls in use. The memorandum entitled, *Inputs to the Pulp and Paper Industry October 2011 Residual Risk Modeling* includes a detailed discussion on the development of the MACT-allowable emissions (available in the docket for this rule making).

3 Risk Results for the Pulp & Paper Source Category

3.1 Source Category Description and Results

The pulp and paper source category includes any facility engaged in the production of pulp and/or paper. This category includes, but is not limited to, integrated mills (where pulp and paper or paperboard are manufactured on-site), non-integrated mills (where paper/paperboard or pulp are manufactured, but not both), and secondary fiber mills (where waste paper is used as the primary raw material. The pulp and paper production process include operations such as pulping, bleaching, chemical recovery, and papermaking. Pulping methods include chemical processes such as kraft, soda, sulfite, and semi-chemical, and mechanical, secondary fiber, or non-wood processes. The MACT standards for the pulp and paper production source category were developed in three parts. This source-category-level risk assessment address the emissions sources covered by the MACT I and MACT III standards[28]. Emission sources regulated under the pulp and paper MACT I standard include all HAP emissions in the kraft, soda, sulfite, and stand-alone semi-chemical pulping processes using wood and all HAP emission points in the bleaching systems. Mills that mechanically pulp wood, pulp secondary fiber or non-wood fibers, and any mills that make paper, paper board, or related products from pulp are entities covered by the MACT III standard. HAP sources covered by the MACT III standard include emission points along the bleaching process. Specifically, bleaching emissions points include storage tanks, tower vents, washer bents, filtrate tank vents, and scrubber outlets. HAP sources covered by the MACT III standard also include paper manufacturing machines and their components (e.g., vacuum pump, storage tank, exhaust). A separate MACT standard[29] applicable to chemical recovery processes at kraft, soda, sulfite, and stand-alone semi-chemical pulp mills was promulgated at a later date. The emissions from the sources covered by this later standard are included in the facility-wide risk analysis. A complete description of the pulp and paper production source category can be found in the text of the NPRM.

We currently estimate that there are 171 pulp and paper facilities operating in the U.S. The ICR data set contains all 171 facilities identified with a pulp and paper production MACT code in the 2005 NEI (updated with the 2010 ICR data). All 171 of these facilities are identified as major sources in the NEI.

The emissions for the pulp and paper source category data set (of 171 facilities) are summarized in Table 3.1-1. The total HAP emissions for the source category are approximately 45,000 tons per year. Based on these data, the HAP emitted in the largest quantities are methanol and acetaldehyde. Emissions of these two HAP make up 91 percent

[28] 40 CFR 63, subpart S: National Emission Standards for Hazardous Air Pollutants from the Pulp and Paper Industry.
[29] 40 CFR Part 63, Subpart MM: National Emission Standards for Hazardous Air Pollutants for Chemical Recovery Combustion Sources at Kraft, Soda, Sulfite, and Stand-Alone Semichemical Pulp Mills

of the total emissions by mass. Persistent and bioaccumulative HAP (PB-HAP) [30] reported as emissions from these facilities include lead, cadmium, mercury, and POM.

Table 3.1-1 Summary of Emissions from the Pulp & Paper Source Category Used in the Residual Risk Assessment and Availability of Dose-Response Values

HAP[a]	Emissions (tpy)	Number of Facilities Reporting HAP (171 facilities in data set)	Prioritized Inhalation Dose-Response Value Identified by OAQPS[b]			PB-HAP?
			Unit Risk Estimate for Cancer?	Reference Concentration for Noncancer?	Health Benchmark Values for Acute Noncancer?	
Methanol	38,650	165		Y	Y	
Acetaldehyde	2,029	160	Y	Y	Y	
Phenol	454	127		Y	Y	
Cresols (mixed)	439	84		Y		
Chloroform	356	128		Y	Y	
o-Cresol	315	41		Y		
Formaldehyde	274	151	Y	Y	Y	
Hydrochloric acid (hydrogen chloride)	259	55		Y	Y	
Biphenyl	218	92			Y	
Hexachloroethane	207	34	Y	Y		
Propionaldehyde	135	106		Y	Y	
1,2,4-Trichlorobenzene	129	94		Y		
Methylene chloride	120	112	Y	Y	Y	
Xylenes (mixed)	98	107		Y	Y	
Carbon disulfide	90	96		Y	Y	
Cumene	83	95		Y	Y	
Toluene	82	126		Y	Y	
Styrene	77	112		Y	Y	
Tetrachloroethene	75	98	Y	Y	Y	
Methyl isobutyl ketone	61	104		Y		
Acetophenone	60	39				
Hexane	56	111		Y	Y	
Carbon tetrachloride	40	92	Y	Y	Y	
Trichloroethylene	37	93	Y	Y	Y	
o-Xylene	36	64		Y	Y	
Benzene	25	128	Y	Y	Y	
Naphthalene	24	105	Y	Y		
Chlorine	24	53		Y	Y	
1,1,2-Trichloroethane	23	88	Y	Y		
1,1,1-Trichloroethane (methyl chloroform)	22	85		Y	Y	
Ethyl benzene	18	76	Y	Y	Y	

[30] Persistent and bioaccumulative HAP are defined in the EPA's *Air Toxics Risk Assessment Library*, Volume 1, EPA-453K-04-001A, as referenced in the ANPRM and provided on the EPA's Technology Transfer Network website for Fate, Exposure, and Risk Assessment at http://www.epa.gov/ttn/fera/risk_atra_vol1.html.

Table 3.1-1 Summary of Emissions from the Pulp & Paper Source Category Used in the Residual Risk Assessment and Availability of Dose-Response Values

HAP[a]	Emissions (tpy)	Number of Facilities Reporting HAP (171 facilities in data set)	Prioritized Inhalation Dose-Response Value Identified by OAQPS[b]			PB-HAP?
			Unit Risk Estimate for Cancer?	Reference Concentration for Noncancer?	Health Benchmark Values for Acute Noncancer?	
Vinyl acetate	16	6		Y	Y	
Chlorobenzene	15	82		Y	Y	
Methyl chloride	14	63		Y	Y	
Hexachlorocyclopentadiene	10	6		Y		
Vinylidene chloride	8	3		Y		
Glycol Ethers						
1,2-Dimethoxyethane	7	31		Y	Y	
Ethylene glycol ethyl ether	0.01	2		Y	Y	
Methly cellosolve acrylate	0.001	1		Y	Y	
Butyl carbitol acetate	0.0002	1		Y	Y	
Acetonitrile	5	2		Y	Y	
Ethylene dichloride	4	75	Y	Y	Y	
Vinyl chloride	4	34	Y	Y	Y	
Triethylamine	3	2		Y	Y	
m-Cresol	3	4		Y		
Carbonyl sulfide	3	18		Y[d]	Y	
Acrylonitrile	3	4	Y	Y	Y	
m-Xylene	2	11		Y	Y	
Chloroacetic acid	2	2			Y	
p-Xylene	1	7		Y	Y	
Pentachlorophenol	1	2	Y	Y		
Ethylene glycol	0.8	9		Y		
Diethanolamine	0.8	4		Y		
Hydrogen cyanide	0.6	1		Y	Y	
1,3-Butadiene	0.5	59	Y	Y	Y	
2,4,5-Trichlorophenol	0.4	1				
p-Cresol	0.3	3		Y		
Methyl bromide	0.3	25		Y	Y	
Nickel compounds	0.2	29	Y	Y	Y	
Acrylamide	0.2	2	Y	Y		
Ethylene dibromide	0.1	20	Y	Y	Y	
Cyanide compounds	0.1	1		Y		
Nitrobenzene	0.09	2	Y	Y		
Antimony compounds	0.08	1		Y		
Lead compounds	0.05	28		Y		Y
Propylene dichloride	0.05	3	Y	Y		
2-Nitropropane	0.05	2	Y	Y		
2,4-Toluene diamine	0.02	3	Y			
Methyl iodide	0.02	3			Y	

Table 3.1-1 Summary of Emissions from the Pulp & Paper Source Category Used in the Residual Risk Assessment and Availability of Dose-Response Values

HAP[a]	Emissions (tpy)	Number of Facilities Reporting HAP (171 facilities in data set)	Prioritized Inhalation Dose-Response Value Identified by OAQPS[b]			PB-HAP?
			Unit Risk Estimate for Cancer?	Reference Concentration for Noncancer?	Health Benchmark Values for Acute Noncancer?	
Chromium Compounds						
Chromium (III) compounds	0.02	27				
Chromium (VI) compounds	0.0007	25	Y	Y		
Ethylidene dichloride (1,1-Dichloroethane)	0.01	3	Y	Y		
Bromoform	0.01	2	Y			
Cadmium compounds	0.01	28	Y	Y		Y
Beryllium compounds	0.01	8	Y	Y	Y	
2,4,6-Trichlorophenol	0.009	2	Y			
Polycyclic Organic Matter (POM)						
POM 72002	0.008	29	Y			Y
Dibenzo(a,h)anthracene	0.0007	7	Y			Y
Indeno(1,2,3-c,d)pyrene	0.000003	7	Y			Y
7,12-Dimethylbenz(a)anthracene	0.000002	3	Y			Y
Benzo(b)fluoranthene	0.0000006	7	Y			Y
Chrysene	0.0000003	5	Y			Y
Benz(a)anthracene	0.0000002	6	Y			Y
Benzo(k)fluoranthene	0.0000002	5	Y			Y
POM 71002	0.0000002	1	Y			Y
3-Methylcholanthrene	0.0000002	3	Y			Y
Benzo(a)pyrene	0.0000002	6	Y			Y
Manganese compounds	0.006	29		Y		
Catechol	0.006	5				
n,n-Dimethylaniline	0.005	1				
Arsenic compounds	0.004	28	Y	Y	Y	
Selenium compounds	0.004	9		Y		
Dibutylphthalate	0.003	5				
1,4-Dichlorobenzene	0.003	7	Y	Y		
Chlorobenzilate	0.002	1	Y			
Cobalt compounds	0.002	24		Y		
Mercury Compounds						
Mercury (elemental)	0.002	27		Y	Y	Y
Mercuric chloride	0.002	27		Y		Y
Bis(2-ethylhexyl)phthalate	0.001	2	Y	Y		
1,1,2,2-Tetrachloroethane	0.0007	1	Y			
Ethylene oxide	0.0003	1	Y	Y	Y	
Aniline	0.00001	3	Y	Y	Y	

[a] Notes for how HAP were speciated for risk assessment:

- For most metals, emissions reported as the elemental metal are combined with metal compound emissions (e.g., "cadmium" emissions modeled as "cadmium & compounds"). In the absence of speciation information, we assume the reported mass is 100 percent metal.
- For emissions reported generically as "chromium" or "chromium & compound," emissions are speciated "chromium (III) compounds" and "chromium (VI) compounds" according to the individual emitting process speciation profile for this source category. Chromium speciation profiles can be found on the EPA's Technology Transfer Network website for emissions inventories at http://www.epa.gov/ttn/chief/net/2005inventory.html. Further information on the development of the chromium speciation profiles used in this assessment can be found in the memorandum entitled, *Inputs to the Pulp and Paper Industry October 2011 Residual Risk Modeling*, found in the docket.
- For emissions reported generically as "mercury" or "mercury & compounds," emissions are speciated for this category as "mercury (elemental)" and "mercuric chloride." Mercury speciation profiles can be found on the EPA's Technology Transfer Network website for emissions inventories at http://www.epa.gov/ttn/chief/net/2005inventory.html.
- For emissions of any chemicals or chemical groups classified as POM, emissions were grouped into POM subgroups as found on EPA's Technology Transfer Network website for the 2005 National-Scale Air Toxics Assessment at http://www.epa.gov/ttn/atw/nata2005/methods.html#pom (*Approach to Modeling POM)*.
- For emissions reported generically as "Glycol Ethers" or specific glycol ethers not found on EPA's Technology Transfer Network for air toxics (see footnote b), emissions were treated as ethylene glycol methyl ether.

[b] Specific dose-response values for each chemical are identified on EPA's Technology Transfer Network website for air toxics at http://www.epa.gov/ttn/atw/toxsource/summary.html.

[c] There is no reference concentration for lead. In considering noncancer hazards for lead in this assessment, we compared rolling three-month average exposure estimates to the National Ambient Air Quality Standard (NAAQS) for lead (0.15 $\mu g/m^3$). These NAAQS for lead were recently reviewed with revisions adopted in October 2008 (http://www.epa.gov/air/lead/actions.html). The primary (health-based) standard is a maximum or not-to-be-exceeded, rolling three-month average, measured as total suspended particles (TSP). The secondary (welfare-based) standard is identical to the primary standard.

[d] A chronic screening level of 0.163 mg/m^3 was developed for carbonyl sulfide by EPA ORD from a No Observed Adverse Effects Level of 200 ppm based on brain lesions and neurophysiological alteration in rodents.

3.2 Risk Characterization

This section presents the results of the risk assessment for the pulp and paper source category. The basic chronic inhalation risk estimates presented here are the maximum individual lifetime cancer risk, the maximum chronic hazard index, and the cancer incidence. We also present results from our acute inhalation impact screening assessment in the form of maximum hazard quotients, as well as the results of our preliminary screen for potential non-inhalation risks from PB-HAP. Also presented are the HAP "drivers," which are the HAP that collectively contribute 90 percent of the maximum cancer risk or maximum hazard at the highest exposure location, as well as a summary of the results of our facility-wide assessments and our analysis of risks associated with the maximum allowed emissions under the current MACT standards. A detailed summary of the facility-specific risk assessment results is available in Appendix 5.

Tables 3.2-1 and 3.2-2 summarize the chronic and acute inhalation risk results for the pulp and paper source category. The results indicate that maximum lifetime individual cancer risks could be up to 10 in a million. The major contributors to this risk are hexachloroethane and naphthalene from kraft processes such as pulp storage, wastewater, and bleaching. The total estimated cancer incidence from this source category is 0.01 excess cancer cases per year, or one excess case in every 100 years. Approximately 40 people are estimated to have cancer

risks at or above 10 in a million, and approximately 76,000 people are estimated to have cancer risks at or above 1 in a million as a result of the emissions from 68 facilities. The maximum chronic noncancer target organ specific hazard index (TOSHI) value for the source category could be up to 0.4 associated with emissions of acetaldehyde, indicating no significant potential for chronic noncancer impacts.

Analysis of potential differences between actual emissions levels and the maximum emissions allowable under the MACT standards were also calculated for stack emissions for this source category. Risk estimates based on the maximum emissions allowable under the MACT standards were calculated from stack emissions obtained from the ICR. Risk results from the inhalation risk assessment indicate that the maximum lifetime individual cancer risk could be up to 10 in a million, and that the maximum chronic noncancer TOSHI value could be up to 0.6 at the MACT-allowable emissions level.

Worst-case acute hazard quotients (HQs) were calculated for every HAP that has an acute benchmark. For cases where the screening acute HQ was greater than 1, we further refined the estimates by determining the highest HQ value that is outside facility boundaries. The highest refined worst-case acute HQ value is 20 (based on the acute REL for acetaldehyde) as shown in Table 3.2-1. The HQ of 20 represents an upper-bound risk estimate and is located in a rural location in which public access is limited or may represent an off-site area that is owned by the facility. An acute noncancer HQ of 3 for this facility would represent an area in which the public has access via a public road. The next highest acute noncancer HQ for this source category would be 6 for chloroform. Nine facilities have estimated acute noncancer HQ values greater than 1, but less than or equal to 6. Based on maximum hourly emission estimates available by emission process group, an emissions multiplier of 2 was used to estimate the peak hourly emission rates for source category. See the memorandum entitled, *Inputs to the Pulp and Paper Industry October 2011 Residual Risk Modeling* for detailed description of how the maximum hourly emissions were developed for this source category (found in the docket for this rule making). Table 3.2-2 provides more information on the refined acute risk estimates for HAP that had an acute HQ greater than 1 for any benchmark.

To better characterize the potential health risks associated with estimated worst-case acute exposures to HAP, and in response to a key recommendation from the Science Advisory Board's peer review of EPA's RTR risk assessment methodologies[31], we examine a wider range of available acute health metrics than we do for our chronic risk assessments. This is in response to the acknowledgement that there are generally more data gaps and inconsistencies in acute reference values than there are in chronic reference values. By definition, the acute CA-REL represents a health-protective level of exposure, with no risk anticipated below those levels, even for repeated exposures; however, the health risk from higher-level exposures is unknown. Therefore, when a CA-REL is exceeded and an AEGL-1 or ERPG-1 level is available (i.e., levels at which mild effects are anticipated in the general public for a single exposure), we have used them as a second comparative measure. Historically, comparisons of the estimated maximum off-site one-hour exposure levels have not been typically made to

[31] The SAB peer review of RTR Risk Assessment Methodologies is available at: http://yosemite.epa.gov/sab/sabproduct.nsf/4AB3966E263D943A8525771F00668381/$File/EPA-SAB-10-007-unsigned.pdf

occupational levels for the purpose of characterizing public health risks in RTR assessments. This is because occupational ceiling values are not generally considered protective for the general public since they are designed to protect the worker population (presumed healthy adults) for short duration (<15 minute) increases in exposure[32]. As a result, for most chemicals, the 15-minute occupational ceiling values are set at levels higher than a one-hour AEGL-1, making comparisons to them irrelevant unless the AEGL-1 or ERPG-1 levels are exceeded. Such is not the case when comparing the available acute inhalation health effect reference values for formaldehyde.

The worst-case maximum estimated 1-hour exposure to formaldehyde outside the facility fence line for the pulp and paper source category is 0.25 mg/m^3. This estimated worst-case exposure exceeds the 1-hour REL by a factor of 5 (HQ_{REL} = 5) and is below the 1-hour AEGL-1 (HQ_{AEGL-1} = 0.2). This exposure estimate is below the AEGL-1, and exceed the workplace ceiling level guideline for the formaldehyde value developed by National Institutes for Occupational Safety and Health (NIOSH)[33] "for any 15 minute period in a work day" (NIOSH REL-ceiling value of 0.12 mg/m^3; HQ_{NIOSH} = 2). The estimate is at the value developed by the American Conference of Governmental Industrial Hygienists (ACGIH)[34] as "not to be exceeded at any time" (ACGIH TLV-ceiling value of 0.37 mg/m^3; HQ_{ACGIH} = 1). Additionally, the estimated maximum acute exposure exceeds the Air Quality Guideline value that was developed by the World Health Organization[35] for 30-minute exposures (0.1 mg/m^3; HQ_{WHO} = 2.5).

To identify potential multipathway health risks from PB-HAP other than lead, we first performed a screening analysis that compared emissions of PB-HAP emitted from the pulp and paper source category to screening emission rates (see section 2.5). The PB-HAP emitted by facilities in this category include cadmium, mercury, and POM (as benzo(a)pyrene toxicity equivalents). Thirty-eight facilities in the source category reported emissions of one or more of these PB-HAP. At the time of proposal for this rulemaking, for all 38 facilities the emissions rate for each PB-HAP was below the screening thresholds, with the exception of one facility's emission rate of POM which exceeded the screening threshold by 2 times. For POM, exceeding the screening emission rate corresponds to a potential for creating a cancer risk in excess of 1 in a million. Since proposal, EPA has refined the emission screening thresholds in the multipathway analysis to use improved toxicity rating and scaling methods for POM and dioxin congeners as well as improved fate, transport, and uptake behavior

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[32] U.S. EPA. (2009) Chapter 2.9 Chemical Specific Reference Values for Formaldehyde in Graphical Arrays of Chemical-Specific Health Effect Reference Values for Inhalation Exposures (Final Report). U.S. Environmental Protection Agency, Washington, DC, EPA/600/R-09/061, and available on-line at http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=211003

[33] National Institutes for Occupational Safety and Health (NIOSH). Occupational Safety and Health Guideline for Formaldehyde; http://www.cdc.gov/niosh/docs/81-123/pdfs/0293.pdf

[34] ACGIH (2001) Formaldehyde. In Documentation of the TLVs® and BEIs® with Other Worldwide Occupational Exposure Values. ACGIH, 1300 Kemper Meadow Drive, Cincinnati, OH 45240 (ISBN: 978-1-882417-74-2) and available on-line at http://www.acgih.org.

[35] WHO (2000). Chapter 5.8 Formaldehyde, in Air Quality Guidelines for Europe, second edition. World Health Organization Regional Publications, European Series, No. 91. Copenhagen, Denmark. Available on-line at http://www.euro.who.int/__data/assets/pdf_file/0005/74732/E71922.pdf.

through the aquatic food chain. (See Appendix 4 for a detailed discussion of the changes to the multipathway screening scenario.) Based on the above changes, the facility-level emissions of POM from this source category are now below the screening threshold by a factor of 9.

In evaluating the potential for multipathway effects from emissions of lead, modeled maximum annual lead concentrations were compared to the NAAQS value for lead (0.15 $\mu g/m^3$), which takes into account multipathway exposures, so a separate multipathway screening value was not developed. Since none of our maximum estimated annual lead concentrations were even close to the NAAQS, we do not expect any significant multipathway exposure and risk due to lead emissions from these facilities.

Table 3.2-1 Summary of Source Category Level Inhalation Risks for Pulp and Paper

Result		HAP "Drivers"
Facilities in Source Category		
Number of Facilities Estimated to be in Source Category	171	n/a
Number of Facilities Identified in the NEI and Modeled in Preliminary Risk Assessment	171	n/a
Cancer Risks		
Maximum Individual Lifetime Cancer Risk (in 1 million)	10	hexachloroethane, naphthalene
Number of Facilities with Maximum Individual Lifetime Cancer Risk:		
Greater than or equal to 100 in 1 million	0	n/a
Greater than or equal to 10 in 1 million	2	hexachloroethane
Greater than or equal to 1 in 1 million	6	hexachloroethane, naphthalene
Chronic Noncancer Risks		
Maximum Respiratory Hazard Index	0.4	acetaldehyde
Number of Facilities with Maximum Respiratory Hazard Index:		
Greater than 100	0	n/a
Greater than 10	0	n/a
Greater than 1	0	n/a
Acute Noncancer Refined Results		
Maximum Acute Hazard Quotient	20 6 5 2	acetaldehyde (REL) chloroform (REL) formaldehyde (REL) methanol (REL)
Number of Facilities With Potential for Acute Effects	9	acetaldehyde, chloroform, formaldehyde, methanol
Population Exposure		
Number of People Living Within 50 Kilometers of Facilities Modeled	50,000,000	n/a
Number of People Exposed to Cancer Risk:		
Greater than or equal to 100 in 1 million	0	n/a
Greater than or equal to 10 in 1 million	40	n/a
Greater than or equal to 1 in 1 million	76,000	n/a
Number of People Exposed to Noncancer Respiratory Hazard Index:		

Table 3.2-1 Summary of Source Category Level Inhalation Risks for Pulp and Paper

Result		HAP "Drivers"
Greater than 100	0	n/a
Greater than 10	0	n/a
Greater than 1	0	n/a
Estimated Cancer Incidence (excess cancer cases per year)	0.01[36]	n/a
Contribution of HAP to Cancer Incidence:		
acetaldehyde	38%	n/a
formaldehyde	36%	n/a
hexachloroethane	9%	n/a
naphthalene	4%	n/a
tetrachloroethene	3%	n/a
1,1,2-trichloroethane	2%	n/a

Table 3.2-2 Summary of Refined Acute Results for Pulp & Paper Facilities

Refined Results		MAXIMUM ACUTE HAZARD QUOTIENTS			ACUTE DOSE-RESPONSE VALUES				
HAP	Max. 1-hr. Air Conc. (mg/m^3)	Based on REL	Based on AEGL-1/ ERPG-1	Based on AEGL-2/ ERPG-2	REL (mg/m^3)	AEGL-1 (1-hr) (mg/m^3)	ERPG-1 (mg/m^3)	AEGL-2 (1-hr) (mg/m^3)	ERPG-2 (mg/m^3)
acetaldehyde	7.1	20	0.09/0.4	0.01/0.02	0.47	81	18	490	360
chloroform	0.9	6		0.003/0.004	0.15			310	240
formaldehyde	0.25	5	0.2/0.2	0.01/0.02	0.055	1.1	1.2	17	12
methanol	64	2	0.09/0.2	0.02/0.05	28	690	260	2700	1300

Notes on Refined Process:

1) The screening was performed for all emitted HAP with available acute dose-response values. Only those pollutants whose screening HQs were greater than 1 for at least one acute threshold value are shown in the table.
2) HAP with available acute dose-response values which are not in the table do not carry any potential for posing acute health risks, based on an analysis of currently available emissions data.

Notes on Acute Dose-Response Values:

REL – California EPA reference exposure level for no adverse effects. Most, but not all, RELs are for 1-hour exposures.

AEGL – Acute exposure guideline levels represent emergency exposure (1-hour) limits for the general public.
AEGL-1 is the exposure level above which it is predicted that the general population, including susceptible individuals, could experience effects that are notable discomfort, but which are transient and reversible upon cessation of exposure.
AEGL-2 is the exposure level above which it is predicted that the general population, including susceptible individuals, could experience irreversible or other serious, long-lasting adverse health effects of an impaired ability to escape.

EPRG – Emergency Removal Program guidelines represent emergency exposure (1-hour) limits for the general public.
ERPG-1 is the maximum level below which it is believed that nearly all individuals could be exposed for up to 1 hour without experiencing other than mild, transient adverse health effects.

[36] We note that the MIR for this source category would not change if the CIIT URE for formaldehyde had been used in the assessment; however, the total cancer incidence would decrease by about 36%. There is an ongoing IRIS reassessment for formaldehyde, and future RTR risk assessments will use the cancer potency for formaldehyde that results from that reassessment. As a result, the current results many not match those of future assessments.

ERPG-2 is the maximum exposure below which it is believed that nearly all individuals could be exposed for up to 1 hour without experiencing or developing irreversible or other serious health effects or symptoms which could impair an individual's ability to take protective action.

The facility-wide MIR and TOSHI, available in Appendix 5, are based on emissions, as collected in the ICR, from all emissions sources at the identified facilities. The results of the facility-wide assessment are summarized in Table 3.2-3. The results indicate that 100 facilities with pulp and paper production processes have facility-wide cancer MIR greater than or equal to 1 in a million. The maximum facility-wide MIR is 30 in a million, with pulp and paper source category contributing 27 percent of the risk. The remaining 63 percent is driven by emissions of arsenic and chromium (VI) from hazardous waste incineration. The maximum facility-wide TOSHI is 2, driven by emissions of antimony compounds from smelt dissolving tank kraft process units. The source category contributes approximately 11 percent to the facility-wide TOSHI.

Table 3.2-3 Source Category Contribution to Facility-Wide Cancer Risks

Polystyrene Production	**Number of Facilities Binned by Facility-Wide MIR (in 1 million)**				
Source Category MIR Contribution to Facility-Wide MIR	<1	1≤ MIR<10	10≤ MIR<100	≥ 100	**Total**
> 90%	19	14	1	0	34
50-90%	21	42	1	0	64
10-50%	27	31	3	0	61
< 10%	4	5	3	0	12
Total	71	92	8	0	171

4 General Discussion of Uncertainties and How They Have Been Addressed

4.1 Exposure Modeling Uncertainties

Although every effort has been made to identify all the relevant facilities and emission points, as well as to develop accurate estimates of the annual emission rates for all relevant HAP, the uncertainties in our emission inventory likely dominate the uncertainties in our exposure estimates. The chronic ambient modeling uncertainties are considered relatively small in comparison, since we are using EPA's refined local dispersion model with site-specific parameters and reasonably representative meteorology. If anything, the population exposure estimates are biased high by not accounting for short- or long-term population mobility, and by neglecting processes like deposition, plume depletion, and atmospheric degradation. Additionally, estimates of the maximum individual risk (MIR) contain uncertainty, because they are derived at census block centroid locations rather than actual residences. This uncertainty is known to create potential underestimates and overestimates of the actual MIR values for individual facilities, but, overall, it is not thought to have a significant impact on

the estimated MIR for a source category. Finally, we did not factor in the possibility of a source closure occurring during the 70-year chronic exposure period, leading to a potential upward bias in both the MIR and population risk estimates; nor did we factor in the possibility of population growth during the 70-year chronic exposure period, leading to a potential downward bias in both the MIR and population risk estimates.

A sensitivity analysis performed for the 1999 NATA found that the selection of the meteorology dataset location could result in a range of chronic ambient concentrations which varied from as much as 17 percent below the predicted value to as much as 84 percent higher than the predicted value. This variability translates directly to the predicted exposures and risks in our assessment, indicating that the actual risks could vary from 17 percent lower to 84 percent higher than the predicted values.

We have purposely biased the acute screening results high, considering that they depend upon the joint occurrence of independent factors, such as hourly emissions rates, meteorology and human activity patterns. Furthermore, in cases where multiple acute threshold values are considered scientifically acceptable we have chosen the most conservative of these threshold values, erring on the side of overestimating potential health risks from acute exposures. In the cases where these results indicated the potential for exceeding short-term health thresholds, we have refined our assessment by developing a better understanding of the geography of the facility relative to potential exposure locations and the true variability of short-term emission rates.

4.2 Uncertainties in the Dose-Response Relationships

In the sections that follow, separate discussions are provided on uncertainty associated with cancer potency factors and for noncancer reference values. Cancer potency values are derived for chronic (lifetime) exposures. Noncancer reference values are generally derived for chronic exposures (up to a lifetime), but may also be derived for acute (<24 hours), short-term (>24 hours up to 30 days), and subchronic (>30 days up to 10 percent of lifetime) exposure durations, all of which are derived based on an assumption of continuous exposure throughout the duration specified. For the purposes of assessing all potential health risks associated with the emissions included in an assessment, we rely on both chronic (cancer and noncancer) and acute (noncancer) benchmarks, which are described in more detail below.

Although every effort is made to identify peer-reviewed dose-response values for all HAPs emitted by the source category included in an assessment, some HAP have no peer-reviewed cancer potency values or reference values for chronic non-cancer or acute effects (inhalation or ingestion). Since exposures to these pollutants cannot be included in a quantitative risk estimate, an understatement of risk for these pollutants at environmental exposure levels is possible.

Additionally, chronic dose-response values for certain compounds included in the assessment may be under EPA IRIS review and revised assessments may determine that these pollutants are more or less potent than currently thought. We will re-evaluate risks if, as a result of these

reviews, a dose-response metric changes enough to indicate that the risk assessment may significantly mischaracterize human health risk

Cancer assessment

The discussion of dose-response uncertainties in the estimation of cancer risk below focuses on the uncertainties associated with the specific approach currently used by the EPA to develop cancer potency factors. In general, these same uncertainties attend the development of cancer potency factors by CalEPA, the source of peer-reviewed cancer potency factors used where EPA-developed values are not yet available. To place this discussion in context, we provide a quote from the EPA's *Guidelines for Carcinogen Risk Assessment* [*30*] (herein referred to as *Cancer Guidelines*). "The primary goal of EPA actions is protection of human health; accordingly, as an Agency policy, risk assessment procedures, including default options that are used in the absence of scientific data to the contrary, should be health protective." The approach adopted in this document is consistent with this approach as described in the *Cancer Guidelines*.

For cancer endpoints EPA usually derives an oral slope factor for ingestion and a unit risk value for inhalation exposures. These values allow estimation of a lifetime probability of developing cancer given long-term exposures to the pollutant. Depending on the pollutant being evaluated, EPA relies on both animal bioassay and epidemiological studies to characterize cancer risk. As a science policy approach, consistent with the *Cancer Guidelines*, EPA uses animal cancer bioassays as indicators of potential human health risk when other human cancer risk data are unavailable.

Extrapolation of study data to estimate potential risks to human populations is based upon EPA's assessment of the scientific database for a pollutant using EPA's guidance documents and other peer-reviewed methodologies. The EPA *Cancer Guidelines* describes the Agency's recommendations for methodologies for cancer risk assessment. EPA believes that cancer risk estimates developed following the procedures described in the *Cancer Guidelines* and outlined below generally provide an upper bound estimate of risk. That is, EPA's upper bound estimates represent a "plausible upper limit to the true value of a quantity" (although this is usually not a true statistical confidence limit).[37] In some circumstances, the true risk could be as low as zero; however, in other circumstances the risk could also be greater.[38] When developing an upper bound estimate of risk and to provide risk values that do not underestimate risk, EPA generally relies on conservative default approaches.[39] EPA also

[37] IRIS glossary (www.epa.gov/NCEA/iris/help_gloss.htm).

[38] The exception to this is the URE for benzene, which is considered to cover a range of values, each end of which is considered to be equally plausible, and which is based on maximum likelihood estimates.

[39] According to the NRC report Science and Judgment in Risk Assessment (NRC, 1994) "[Default] options are generic approaches, based on general scientific knowledge and policy judgment, that are applied to various elements of the risk-assessment process when the correct scientific model is unknown or uncertain." The 1983 NRC report Risk Assessment in the Federal Government: Managing the Process defined default option as "the option chosen on the basis of risk assessment policy that appears to be the best choice in the absence of data to the contrary" (NRC, 1983a, p. 63). Therefore, default options are not rules that bind the agency; rather, the agency may depart from them in evaluating the risks posed by a specific substance when it believes this to be appropriate. In keeping with EPA's goal of protecting public health and the environment, default assumptions

uses the upper bound (rather than lower bound or central) estimates in its assessments, although it is noted that this approach can have limitations for some uses (e.g. priority setting, expected benefits analysis).

Such health risk assessments have associated uncertainties, some which may be considered quantitatively, and others which generally are expressed qualitatively. Uncertainties may vary substantially among cancer risk assessments associated with exposures to different pollutants, since the assessments employ different databases with different strengths and limitations and the procedures employed may differ in how well they represent actual biological processes for the assessed substance. EPA's *Risk Characterization Handbook* also recommends that risk characterizations present estimates demonstrating the impact on the assessment of alternative choices, data, models and assumptions [*31*]. Some of the major sources of uncertainty and variability in deriving cancer risk values are described more fully below.

(1) The qualitative similarities or differences between tumor responses observed in experimental animal bioassays and those which would occur in humans are a source of uncertainty in cancer risk assessment. In general, EPA does not assume that tumor sites observed in an experimental animal bioassay are necessarily predictive of the sites at which tumors would occur in humans.[40] However, unless scientific support is available to show otherwise, EPA assumes that tumors in animals are relevant in humans, regardless of target organ concordance. For a specific pollutant, qualitative differences in species responses can lead to either under-estimation or over-estimation of human cancer risks.

(2) Uncertainties regarding the most appropriate dose metric for an assessment can also lead to differences in risk predictions. For example, the measure of dose is commonly expressed in units of mg/kg/d ingested or the inhaled concentration of the pollutant. However, data may support development of a pharmacokinetic model for the absorption, distribution, metabolism and excretion of an agent, which may result in improved dose metrics (e.g., average blood concentration of the pollutant or the quantity of agent metabolized in the body). Quantitative uncertainties result when the appropriate choice of a dose metric is uncertain or when dose metric estimates are themselves uncertain (e.g., as can occur when alternative pharmacokinetic models are available for a compound). Uncertainty in dose estimates may lead to either over or underestimation of risk.

(3) For the quantitative extrapolation of cancer risk estimates from experimental animals to humans, EPA uses scaling methodologies (relating expected response to differences in physical size of the species), which introduce another source of uncertainty. These methodologies are based on both biological data on differences in rates of process according to species size and empirical comparisons of toxicity between experimental animals and humans. For a particular pollutant, the quantitative difference in cancer potency between

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are used to ensure that risk to chemicals is not underestimated (although defaults are not intended to overtly overestimate risk). See EPA 2004 An Examination of EPA Risk Assessment Principles and Practices, EPA/100/B-04/001 available at: http://www.epa.gov/osa/pdfs/ratf-final.pdf.

[40] Per the EPA Cancer Guidelines: "The default option is that positive effects in animal cancer studies indicate that the agent under study can have carcinogenic potential in humans." and "Target organ concordance is not a prerequisite for evaluating the implications of animal study results for humans."

experimental animals and humans may be either greater than or less than that estimated by baseline scientific scaling predictions due to uncertainties associated with limitations in the test data and the correctness of scaled estimates.

(4) EPA cancer risk estimates, whether based on epidemiological or experimental animal data, are generally developed using a benchmark dose (BMD) analysis to estimate a dose at which there is a specified excess risk of cancer, which is used as the point of departure (or POD) for the remainder of the calculation. Statistical uncertainty in developing a POD using a benchmark dose (BMD) approach is generally addressed though use of the 95 percent lower confidence limit on the dose at which the specified excess risk occurs (the BMDL), decreasing the likelihood of understating risk. EPA has generally utilized the multistage model for estimation of the BMDL using cancer bioassay data (see further discussion below).

(5) Extrapolation from high to low doses is an important, and potentially large, source of uncertainty in cancer risk assessment. EPA uses different approaches to low dose risk assessment (i.e., developing estimates of risk for exposures to environmental doses of an agent from observations in experimental or epidemiological studies at higher dose) depending on the available data and understanding of a pollutant's mode of action (i.e., the manner in which a pollutant causes cancer). EPA's *Cancer Guidelines* express a preference for the use of reliable, compound-specific, biologically-based risk models when feasible; however, such models are rarely available. The mode of action for a pollutant (i.e., the manner in which a pollutant causes cancer) is a key consideration in determining how risks should be estimated for low-dose exposure. A reference value is calculated when the available mode of action data show the response to be nonlinear (e.g., as in a threshold response). A linear low-dose (straight line from POD) approach is used when available mode of action data support a linear (e.g., nonthreshold response) or as the most common default approach when a compound's mode of action is unknown. Linear extrapolation can be supported by both pollutant-specific data and broader scientific considerations. For example, EPA's *Cancer Guidelines* generally consider a linear dose-response to be appropriate for pollutants that interact with DNA and induce mutations. Pollutants whose effects are additive to background biological processes in cancer development can also be predicted to have low-dose linear responses, although the slope of this relationship may not be the same as the slope estimated by the straight line approach.

EPA most frequently utilizes a linear low-dose extrapolation approach as a baseline science-policy choice (a "default") when available data do not allow a compound-specific determination. This approach is designed to not underestimate risk in the face of uncertainty and variability. EPA believes that linear dose-response models, when appropriately applied as part of EPA's cancer risk assessment process, provide an upper bound estimate of risk and generally provide a health protective approach. Note that another source of uncertainty is the characterization of low-dose nonlinear, non-threshold relationships. The National Academy of Sciences has encouraged the exploration of sigmoidal type functions (e.g., log-probit models) in representing dose response relationships due to the variability in response within human populations. Another National Research Council (NRC) report [*32*] suggests that models based on distributions of individual thresholds are likely to lead to sigmoidal-shaped dose-response functions for a population. This report notes sources of variability in the

human population: "One might expect these individual tolerances to vary extensively in humans depending on genetics, coincident exposures, nutritional status, and various other susceptibility factors..." Thus, if a distribution of thresholds approach is considered for a carcinogen risk assessment, application would depend on ability of modeling to reflect the degree of variability in response in human populations (as opposed to responses in bioassays with genetically more uniform rodents). Note also that low dose linearity in risk can arise for reasons separate from population variability: due to the nature of a mode of action and additivity of a chemical's effect on top of background chemical exposures and biological processes.

As noted above, EPA's current approach to cancer risk assessment typically utilizes a straight line approach from the BMDL. This is equivalent to using an upper confidence limit on the slope of the straight line extrapolation. The impact of the choice of the BMDL on bottom line risk estimates can be quantified by comparing risk estimates using the BMDL value to central estimate BMD values, although these differences are generally not a large contributor to uncertainty in risk assessment (Subramaniam et. al., 2006) [*33*]. It is important to note that earlier EPA assessments, including the majority of those for which risk values exist today, were generally developed using the multistage model to extrapolate down to environmental dose levels and did not involve the use of a POD. Subramaniam et. al. (2006) also provide comparisons indicating that slopes based on straight line extrapolation from a POD do not show large differences from those based on the upper confidence limit of the multistage model.

(6) Cancer risk estimates do not generally make specific adjustments to reflect the variability in response within the human population — resulting in another source of uncertainty in assessments. In the diverse human population, some individuals are likely to be more sensitive to the action of a carcinogen than the typical individual, although compound-specific data to evaluate this variability are generally not available. There may also be important life stage differences in the quantitative potency of carcinogens and, with the exception of the recommendations in EPA's Supplemental Cancer Guidance for carcinogens with a mutagenic mode of action, risk assessments do not generally quantitatively address life stage differences. However, one approach used commonly in EPA assessments that may help address variability in response is to extrapolate human response from results observed in the most sensitive species and sex tested, resulting typically in the highest URE which can be supported by reliable data, thus supporting estimates that are designed not to underestimate risk in the face of uncertainty and variability.

Chronic noncancer assessment

Chronic noncancer reference values represent chronic exposure levels that are intended to be health-protective. That is, EPA and other organizations which develop noncancer reference values (e.g., the Agency for Toxic Substances and Disease Registry – ATSDR) utilize an approach that is intended not to underestimate risk in the face of uncertainty and variability. When there are gaps in the available information, uncertainty factors (UFs) are applied to derive reference values that are intended to be protective against appreciable risk of

deleterious effects. Uncertainty factors are commonly default values[41] e.g., factors of 10 or 3, used in the absence of compound-specific data; where data are available, uncertainty factors may also be developed using compound-specific information. When data are limited, more assumptions are needed and more default factors are used. Thus there may be a greater tendency to overestimate risk—in the sense that further study might support development of reference values that are higher (i.e., less potent) because fewer default assumptions are needed. However, for some pollutants it is possible that risks may be underestimated.

For non-cancer endpoints related to chronic exposures, EPA derives a Reference Dose (RfD) for exposures via ingestion, and a Reference Concentration (RfC) for inhalation exposures. These values provide an estimate (with uncertainty spanning perhaps an order of magnitude) of daily oral exposure (RfD) or of a continuous inhalation exposure (RfC) to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime.[42] To derive values that are intended to be "without appreciable risk," EPA's methodology relies upon an uncertainty factor (UF) approach [*34*],[*35*] which includes consideration of both uncertainty and variability.

EPA begins by evaluating all of the available peer-reviewed literature to determine non-cancer endpoints of concern, evaluating the quality, strengths and limitations of the available studies. EPA typically chooses the relevant endpoint that occurs at the lowest dose, often using statistical modeling of the available data, and then determines the appropriate point of departure (POD) for derivation of the reference value. A POD is determined by (in order of preference): (1) a statistical estimation using the benchmark dose (BMD) approach; (2) use of the dose or concentration at which the toxic response was not significantly elevated (no observed adverse effect level— NOAEL); or (3) use of the lowest observed adverse effect level (LOAEL).

A series of downward adjustments using default UFs is then applied to the POD to estimate the reference value [*36*]. While collectively termed "UFs", these factors account for a number of different quantitative considerations when utilizing observed animal (usually rodent) or human toxicity data in a risk assessment. The UFs are intended to account for: (1) variation in susceptibility among the members of the human population (i.e., inter-individual variability); (2) uncertainty in extrapolating from experimental animal data to humans (i.e., interspecies differences); (3) uncertainty in extrapolating from data obtained in a study with

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[41] According to the NRC report *Science and Judgment in Risk Assessment* (NRC, 1994) "[Default] options are generic approaches, based on general scientific knowledge and policy judgment, that are applied to various elements of the risk-assessment process when the correct scientific model is unknown or uncertain." The 1983 NRC report, *Risk Assessment in the Federal Government: Managing the Process* defined *default option* as "the option chosen on the basis of risk assessment policy that appears to be the best choice in the absence of data to the contrary" (NRC, 1983a, p. 63). Therefore, default options are not rules that bind the agency; rather, the agency may depart from them in evaluating the risks posed by a specific substance when it believes this to be appropriate. In keeping with EPA's goal of protecting public health and the environment, default assumptions are used to ensure that risk to chemicals is not underestimated (although defaults are not intended to overtly overestimate risk). See EPA 2004 *An examination of EPA Risk Assessment Principles and Practices,* EPA/100/B-04/001 available at: http://www.epa.gov/osa/pdfs/ratf-final.pdf.

[42] See IRIS glossary

less-than-lifetime exposure (i.e., extrapolating from subchronic to chronic exposure); (4) uncertainty in extrapolating from a LOAEL in the absence of a NOAEL; and (5) uncertainty when the database is incomplete or there are problems with applicability of available studies. When scientifically sound, peer-reviewed assessment-specific data are not available, default adjustment values are selected for the individual UFs. For each type of uncertainty (when relevant to the assessment), EPA typically applies an UF value of 10 or 3 with the cumulative UF value leading to a downward adjustment of 10-3000 fold from the selected POD. An UF of 3 is used when the data do not support the use of a 10-fold factor. If an extrapolation step or adjustment is not relevant to an assessment (e.g., if applying human toxicity data and an interspecies extrapolation is not required) the associated UF is not used. The major adjustment steps are described more fully below.

1) Heterogeneity among humans is a key source of variability as well as uncertainty. Uncertainty related to human variation is considered in extrapolating doses from a subset or smaller-sized population, often of one sex or of a narrow range of life stages (typical of occupational epidemiologic studies), to a larger, more diverse population. In the absence of pollutant-specific data on human variation, a 10-fold UF is used to account for uncertainty associated with human variation. Human variation may be larger or smaller; however, data to examine the potential magnitude of human variability are often unavailable. In some situations, a smaller UF of 3 may be applied to reflect a known lack of significant variability among humans.

2) Extrapolation from results of studies in experimental animals to humans is a necessary step for the majority of chemical risk assessments. When interpreting animal data, the concentration at the POD (e.g. NOAEL, BMDL) in an animal model (e.g. rodents) is extrapolated to estimate the human response. While there is long-standing scientific support for the use of animal studies as indicators of potential toxicity to humans, there are uncertainties in such extrapolations. In the absence of data to the contrary, the typical approach is to use the most relevant endpoint from the most sensitive species and the most sensitive sex in assessing risks to the average human. Typically, compound specific data to evaluate relative sensitivity in humans versus rodents are lacking, thus leading to uncertainty in this extrapolation. Size-related differences (allometric relationships) indicate that typically humans are more sensitive than rodents when compared on a mg/kg/day basis. The default choice of 10 for the interspecies UF is consistent with these differences. For a specific chemical, differences in species responses may be greater or less than this value.

Pharmacokinetic models are useful to examine species differences in pharmacokinetic processing and associated uncertainties; however, such dosimetric adjustments are not always possible. Information may not be available to quantitatively assess toxicokinetic or toxicodynamic differences between animals and humans, and in many cases a 10-fold UF (with separate factors of 3 for toxicokinetic and toxicodynamic components) is used to account for expected species differences and associated uncertainty in extrapolating from laboratory animals to humans in the derivation of a reference value. If information on one or the other of these components is available and accounted for in the cross-species extrapolation, a UF of 3 may be used for the remaining component.

3) In the case of reference values for chronic exposures where only data from shorter durations are available (e.g., 90-day subchronic studies in rodents) or when such data are judged more appropriate for development of an RfC, an additional UF of 3 or 10-fold is typically applied unless the available scientific information supports use of a different value.

4) Toxicity data are typically limited as to the dose or exposure levels that have been tested in individual studies; in an animal study, for example, treatment groups may differ in exposure by up to an order of magnitude. The preferred approach to arrive at a POD is to use BMD analysis; however, this approach requires adequate quantitative results for a meaningful analysis, which is not always possible. Use of a NOAEL is the next preferred approach after BMD analysis in determining a POD for deriving a health effect reference value. However, many studies lack a dose or exposure level at which an adverse effect is not observed (i.e., a NOAEL is not identified). When using data limited to a LOAEL, a UF of 10 or 3-fold is often applied.

5) The database UF is intended to account for the potential for deriving an underprotective RfD/RfC due to a data gap preventing complete characterization of the chemical's toxicity. In the absence of studies for a known or suspected endpoint of concern, a UF of 10 or 3-fold is typically applied.

There is no RfD or other comparable chronic health benchmark value for lead compounds. Thus, to address multipathway human health and environmental risks associated with emissions of lead from these facilities, ambient lead concentrations were compared to the NAAQS for lead. In developing the NAAQS for lead, EPA considered human health evidence reporting adverse health effects associated with lead exposure, as well as an EPA conducted multipathway risk assessment that applied models to estimate human exposures to air-related lead and the associated risk (73FR at 66979). EPA also explicitly considered the uncertainties associated with both the human health evidence and the exposure and risk analyses when developing the NAAQS for lead. For example, EPA considered uncertainties in the relationship between ambient air lead and blood lead levels (73FR at 66974), as well as uncertainties between blood lead levels and loss of IQ points in children (73FR at 66981). In considering the evidence and risk analyses and their associated uncertainties, the EPA Administrator noted his view that there is no evidence- or risk-based bright line that indicates a single appropriate level. Instead, he noted, there is a collection of scientific evidence and judgments and other information, including information about the uncertainties inherent in many relevant factors, which needs to be considered together in making this public health policy judgment and in selecting a standard level from a range of reasonable values (73FR at 66998). In so doing, the Administrator decided that, a level for the primary lead standard of 0.15 $\mu g/m^3$, in combination with the specified choice of indicator, averaging time, and form, is requisite to protect public health, including the health of sensitive groups, with an adequate margin of safety (73FR at 67006). A thorough discussion of the health evidence, risk and exposure analyses, and their associated uncertainties can be found in EPA's final rule revising the lead NAAQS (73 FR 66970-66981, November 12, 2008).

We also note the uncertainties associated with the health-based (i.e., primary) NAAQS are likely less than the uncertainties associated with dose-response values developed for many of

the other HAP, particularly those HAP for which no human health data exist. In 1988, EPA's IRIS program reviewed the health effects data regarding lead and its inorganic compounds and determined that it would be inappropriate to develop an RfD for these compounds, saying, "A great deal of information on the health effects of lead has been obtained through decades of medical observation and scientific research. This information has been assessed in the development of air and water quality criteria by the Agency's Office of Health and Environmental Assessment (OHEA) in support of regulatory decision-making by the Office of Air Quality Planning and Standards (OAQPS) and by the Office of Drinking Water (ODW). By comparison to most other environmental toxicants, the degree of uncertainty about the health effects of lead is quite low. It appears that some of these effects, particularly changes in the levels of certain blood enzymes and in aspects of children's neurobehavioral development, may occur at blood lead levels so low as to be essentially without a threshold. The Agency's RfD Work Group discussed inorganic lead (and lead compounds) at two meetings (07/08/1985 and 07/22/1985) and considered it inappropriate to develop an RfD for inorganic lead." EPA's IRIS assessment for Lead and compounds (inorganic) (CASRN 7439-92-1) is available at http://www.epa.gov/iris/subst/0277.htm.

We note further that because of the multi-pathway, multi-media impacts of lead, the risk assessment supporting the NAAQS considered direct inhalation exposures and indirect air-related multi-pathway exposures from industrial sources like primary and secondary lead smelting operations. It also considered background lead exposures from other sources (like contaminated drinking water and exposure to lead-based paints). In revising the NAAQS for lead, we note that the Administrator placed more weight on the evidence-based framework and less weight on the results from the risk assessment, although he did find the risk estimates to be roughly consistent with and generally supportive of the evidence-based framework applied in the NAAQS determination (73FR at 67004). Thus, when revising the NAAQS for lead to protect public health with an adequate margin of safety, EPA considered both the evidence-based framework and the risk assessment, albeit to different extents.

Acute noncancer assessment

Many of the UFs used to account for variability and uncertainty in the development of acute reference values are quite similar to those developed for chronic durations, but more often using individual UF values that may be less than 10. UFs are applied based on chemical-specific or health effect-specific information (e.g., simple irritation effects do not vary appreciably between human individuals, hence a value of 3 is typically used), or based on the purpose for the reference value (see the following paragraph). The UFs applied in acute reference value derivation include: 1) heterogeneity among humans; 2) uncertainty in extrapolating from animals to humans; 3) uncertainty in LOAEL to NOAEL adjustments; and 4) uncertainty in accounting for an incomplete database on toxic effects of potential concern. Additional adjustments are often applied to account for uncertainty in extrapolation from observations at one exposure duration (e.g., 4 hours) to arrive at a POD for derivation of an acute reference value at another exposure duration (e.g., 1 hour).

Not all acute reference values are developed for the same purpose and care must be taken when interpreting the results of an acute assessment of human health effects relative to the

reference value or values being exceeded. Where relevant to the estimated exposures, the lack of threshold values at different levels of severity should be factored into the risk characterization as potential uncertainties.

5 References

1. US EPA. National Emission Standards for Hazardous Air Pollutants. http://www.epa.gov/ttn/atw/mactfnlalph.html.

2. US EPA, 2009. Risk and Technology Review (RTR) Risk Assessment Methodologies: For Review by the EPA's Science Advisory Board with Case Studies – MACT I Petroleum Refining Sources and Portland Cement Manufacturing. EPA-452/R-09-006. http://www.epa.gov/ttn/atw/rrisk/rtrpg.html.

3. US EPA, 2010. SAB's Response to EPA's RTR Risk Assessment Methodologies. http://yosemite.epa.gov/sab/sabproduct.nsf/4AB3966E263D943A8525771F00668381/$File/EPA-SAB-10-007-unsigned.pdf

4. US EPA, 2010. Memorandum from Dave Guinnup to RTR Wood Furniture Docket, entitled, "EPA's Actions in Response to Key Recommendations of the SAB Review of RTR Risk Assessment Methodologies.

5. US EPA, 2005. Revision to the Guideline on Air Quality Models: Adoption of a Preferred General Purpose (Flat and Complex Terrain) Dispersion Model and Other Revisions; Final Rule. 40 CFR Part 51. http://www.epa.gov/EPA-AIR/2005/November/Day-09/a21627.htm

6. US EPA, 2004. Users' guide for the AMS/EPA regulatory model – AERMOD. EPA-454/B-03-001. http://www.epa.gov/scram001/7thconf/aermod/aermodugb.pdf.

7. Allen, D., C. Murphy, Y. Kimura, W. Vizuete, T. Edgar, H. Jeffries, B.-U. Kim, M. Webster, and M. Symons, 2004. Variable industrial VOC emissions and their impact on ozone formation in the Houston Galveston Area. Final Report: Texas Environmental Research Consortium Project H-13. http://files.harc.edu/Projects/AirQuality/Projects/H013.2003/H13FinalReport.pdf

8. US EPA, 2004. Air Toxics Risk Assessment Reference Library, Volume 1. EPA-453-K-04-001A. http://www.epa.gov/ttn/fera/risk_atra_vol1.html.

9. US EPA, 2012. Table 1. Prioritized Chronic Dose-Response Values (5/21/12). Office of Air Quality Planning and Standards. http://www.epa.gov/ttn/atw/toxsource/table1.pdf

10. US EPA, 2005. 1999 National Air Toxics Risk Assessment. http://www.epa.gov/ttn/atw/nata1999

11. US EPA, 2006. Integrated Risk Information System. http://www.epa.gov/iris/index.html.

12. US Agency for Toxic Substances and Disease Registry. 2006. Minimum Risk Levels (MRLs) for Hazardous Substances. http://www.atsdr.cdc.gov/mrls/index.html.

13. US EPA, 1994. US Environmental Protection Agency. Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry. EPA/600/8-90/066F. Office of Research and Development. Washington, DC: U.S.EPA.

14. NRC, 1994. National Research Council. Science and Judgment in Risk Assessment. Washington, DC: National Academy Press.

15. CA Office of Environmental Health Hazard Assessment, 2005. Chronic Reference Exposure Levels Adopted by OEHHA as of December 2008. http://www.oehha.ca.gov/air/chronic_rels/AllChrels.html.

16. CA Office of Environmental Health Hazard Assessment, 2005. Technical Support Document for Describing Available Cancer Potency Factors, May 2005. http://www.oehha.ca.gov/air/hot_spots/may2005tsd.html.

17. US EPA, 2006. Approach for modeling POM. Technical support information for the 1999 National Air Toxics Assessment. http://www.epa.gov/ttn/atw/nata1999/99pdfs/pomapproachjan.pdf.

18. US EPA, 2011. Table 2. Acute Dose-Response Values for Screening Risk Assessments (12/19/2011). Office of Air Quality Planning and Standards. http://www.epa.gov/ttn/atw/toxsource/table2.pdf

19. U.S. EPA, 2009. Graphical Arrays of Chemical-Specific Health Effect Reference Values for Inhalation Exposures [Final Report]. EPA/600/R-09/061, 2009. http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=211003

20. CA Office of Environmental Health Hazard Assessment, 2000. All Acute Reference Exposure Levels developed by OEHHA as of May 2000. http://www.oehha.ca.gov/air/acute_rels/allAcRELs.html

21. American Industrial Hygiene Association, 2011. Current AIHA ERPG Values. http://www.aiha.org/insideaiha/GuidelineDevelopment/ERPG/Documents/2011erpgweelhandbook_table-only.pdf.

22. US EPA, 1995. Guidance for Risk Characterization. Science Policy Council. http://www.epa.gov/OSA/spc/pdfs/rcguide.pdf.

23. US EPA, 2000. Risk Characterization Handbook. EPA 100-B-00-002.

24. US EPA, 2002. EPA's Guidelines for Ensuring and Maximizing the Quality, Objectivity, Utility, and Integrity of Information Disseminated by the Environmental Protection Agency. EPA Office of Environmental Information. EPA/260R-02-008. http://www.epa.gov/quality/informationguidelines/documents/EPA_InfoQualityGuidelines.pdf

25. US EPA, 2006. Performing risk assessments that include carcinogens described in the Supplemental Guidance as having a mutagenic mode of action. Science Policy Council Cancer Guidelines Implementation Workgroup Communication II: Memo from W.H. Farland dated 14 June 2006. http://www.epa.gov/osa/spc/pdfs/CGIWGCommunication_II.pdf

26. US EPA, 2005. Supplemental guidance for assessing early-life exposure to carcinogens. EPA/630/R-03003F. http://www.epa.gov/ttn/atw/childrens_supplement_final.pdf.

27. US EPA, 2005. Science Policy Council Cancer Guidelines Implementation Workgroup Communication I: Memo from W.H. Farland dated 4 October 2005 to Science Policy Council. http://www.epa.gov/osa/spc/pdfs/canguid1.pdf

28. US EPA, 1986. Guidelines for the Health Risk Assessment of Chemical Mixtures. EPA-630-R-98-002. http://www.epa.gov/NCEA/raf/pdfs/chem_mix/chemmix_1986.pdf

29. US EPA, 2000. Supplementary Guidance for Conducting Health Risk Assessment of Chemical Mixtures. EPA-630/R-00-002. http://www.epa.gov/ncea/raf/pdfs/chem_mix/chem_mix_08_2001.pdf.

30. US EPA, 2005. Guidelines for Carcinogen Risk Assessment (2005). U.S. Environmental Protection Agency, Washington, DC, EPA/630/P-03/001F, 2005. http://cfpub.epa.gov/ncea/raf/recordisplay.cfm?deid=116283

31. US EPA, 2000. Risk Characterization Handbook. EPA 100-B-00-002.

32. NRC (National Research Council) 2006. Assessing the Human Health Risks of Trichloroethylene. National Academies Press, Washington DC.

33. R.P. Subramaniam, P. White and V.J. Cogliano. 2006. Comparison of cancer slope factors using different statistical approaches, Risk Anal. Vol. 26, p. 825–830.

34. US EPA. 1993. Reference Dose (RfD): Description and Use in Health Risk Assessments. http://www.epa.gov/iris/rfd.htm

35. US EPA. 1994. Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry. http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=71993.

36. US EPA. 2002. A Review of the Reference Dose and Reference Concentration Processes. http://www.epa.gov/ncea/iris/RFD_FINAL1.pdf